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YELLOW FEVER.

BEEUWKES (Henry) & MAHAFFY (A. F.). **The Past Incidence and Distribution of Yellow Fever in West Africa as indicated by Protection Test Surveys.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1934. June 30. Vol. 28. No. 1. pp. 39–76. With 6 maps & 1 graph. [12 refs.]

This most interesting article summarizes the results of a yellow fever protection test survey comprising the examination of 7,580 sera collected in 181 towns in eight colonies of West Africa, *viz.*, Nigeria, Gold Coast, Sierra Leone, Gambia, Liberia, Dahomey, Niger and the (French) Sudan.

These results confirm and very greatly extend those previously recorded by Beeuwkes, BAUER & Mahaffy [see this *Bulletin*, Vol. 28, p. 283], but in this earlier work monkeys were used as the test animals and consequently only a very limited number of tests could be made. As a result of the introduction of the mouse protection test an almost unlimited extension of the work has become possible.

The usual number of specimens of serum collected in a centre was 25 from children and 25 from adults. The blood was collected in 30 cc. vacuum tubes which were completely filled and whenever possible preserved in refrigerators until tested. Six young mice of a susceptible strain were used for testing each specimen; when all or five mice survived the specimen was considered as positive, when four or more died, as negative, otherwise as inconclusive. The results of these tests are given first in tabular form and then discussed in detail under the headings of the various colonies. The findings again suggest that yellow fever is far more prevalent in West Africa than was previously supposed, for large numbers of positive sera were found both in the coastal regions and in the interior, and approximately 25 per cent. of all specimens examined gave positive results. Practically the whole area covered by the survey was found to be infectible and few towns seem to have escaped the disease during the present generation. Positive sera were obtained from natives even on the Jos Plateau at an elevation of more than 4,000 feet.

In Nigeria the most striking finding is the demonstration of the heavy past incidence of yellow fever compared with the reported cases. During the past 25 years it has been recorded in only 22 places, and most of these were isolated cases among Europeans. In the past 10 years a total of 53 cases have been observed in only 10 towns, and throughout

the whole of Northern Nigeria above 10° latitude only 3 cases of yellow fever have ever been reported. Nevertheless, the results of protection tests suggest that few towns of any size have escaped, and that large numbers of even the smallest villages have been visited by the disease within recent years. Specimens of sera from children under 15 years of age living in 99 towns and villages were examined and only 20 per cent. of these localities were negative. When adults were included only four towns were completely negative. Even in sparsely inhabited areas a large number of specimens were positive, and it is known that epidemics occur in the semi-arid region north of Nigeria. The Sahara desert, however, forms an effective barrier against the infection.

It is evident that yellow fever is rarely recognized in the native, for in a large proportion of the positive localities, with percentages reaching as high as 92 per cent. positive, the disease had never been recorded. On the contrary, the examination of specimens of blood from Europeans who had lived in West African colonies for 10 to 40 years did not reveal any positive sera. It is evident that mild unrecognized cases of the disease, so common in the native, are relatively infrequent in Europeans living in West Africa.

The results of the protection test survey by colonies are summarized in the following table :—

Colony.	Number of towns where specimens were collected.	Total number of specimens.	Number positive.	Per cent. positive.	Remarks.
Nigeria ...	120	5,607	1,508	27	Includes specimens collected in numerous small villages.
Gold Coast	33	861	168	20	
Sierra Leone	5	149	19	13	
Gambia ...	2	68	18	26	Collection at Georgetown included donors from many small villages in the vicinity.
Liberia ...	3	96	6	6	Collections at the Firestone Plantation were from donors coming from many towns in the interior.
Dahomey	5	188	56	30	
Niger ...	11	481	104	22	
Sudan ...	2	130	Nil	Nil	
Total ...	181	7,580	1,879	25	

The findings confirm the opinion that an endemic area of yellow fever exists in South-Western Nigeria, which may extend westward into neighbouring colonies. The meteorological conditions in Northern Nigeria, however, seem to be unfavourable for the permanent existence of the disease. Age-group studies in large towns of endemic areas show that the disease is not confined to children but that the percentage of

immunes gradually increases up to old age, and some persons escape throughout life. It is also shown that extensive epidemics have occurred in endemic areas.

The great value of piped water supplies and effective sanitary services, in reducing or eliminating yellow fever infection, is brought out in many of the places studied and especially in Freetown, Sierra Leone, and in some of the coastal towns of the Gold Coast and Nigeria.

[In the space at our disposal it is only possible to give a brief outline of the contents of this valuable report which should be read in its entirety by all those interested in yellow fever, or in general epidemiology.] *E. Hindle.*

Boyé. Les recherches concernant le test de protection contre la fièvre jaune en Afrique Equatoriale Française et au Cameroun. [**The Protection Test against Yellow Fever in French Equatorial Africa and the Cameroons.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. June. Vol. 26. No. 6. pp. 1061–1066.

A total of 1,008 samples of blood were collected by Burke and Durand from various parts of French Equatorial Africa and the Cameroons, and tested for yellow fever immunity by means of the mouse protection test. The results are given in tabular form and show that out of 32 localities in Equatorial Africa only 5 were negative, the remaining 27 showing percentages of positive sera ranging from a maximum of 95·5 per cent. down to a minimum of 4 per cent., with an average of 42·63 per cent.

These figures are very difficult to understand for they are much higher than the averages obtained in the epidemic centres of French West Africa where there have been great epidemics in the past and annual recurrences of the disease. In Equatorial Africa, Libreville has been occupied for 80 years and yet in spite of an index of 20 per cent. positive, there has never been a clinically diagnosed case of yellow fever in any European. The author calls attention to similar results in other localities, including the positive results recorded in the Bahr-el-Ghazal and Uganda; and points out that these discrepancies could be explained if the mouse protection test was not strictly specific. Consequently it is of the highest importance to know if the presence of seroprotection is a certain indication of previous infection with yellow fever either apparent or non-apparent. *E. H.*

Mouchet (R.), van Hoof (L.), Duren (A.), Fontana (L.), Clarebout (G.), Henry (E.), & Henrard (C.). Résumé de l'enquête sur l'endémicité amarile au Congo Belge en 1932–1933. [**Summary of an Inquiry into the Endemicity of Yellow Fever in the Belgian Congo during 1932 and 1933.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. June. Vol. 26. No. 6. pp. 1067–1071.

Details of this inquiry will be published elsewhere, but the results of mouse protection tests with sera collected from various parts of the Belgian Congo show similar points to those recorded by Boyé [see above], and in particular a high percentage of positive sera in regions where no suspected case of yellow fever has ever been recorded. The epidemic at Matadi seems to be extinct, for whilst the adult natives gave an incidence of 41 per cent. positive, the children showed only 6 per cent. positive. It is curious, however, that regions in the interior

of the Congo where the disease has never been diagnosed clinically, give a higher percentage of positive sera than the coastal regions.

E. H.

JORGE (Ricardo). Sur la prospection biodémique de la fièvre jaune. [**The Biological Survey of Yellow Fever Endemicity.**]—*Bull. Office Internat. d'Hyg Publique.* 1934. Aug. Vol. 26. No. 8. pp. 1396–1402.

The results furnished by Dr. BEEUWKES of the examination of 950 sera for yellow fever immunity from 19 localities in Angola, by means of the mouse protection test, show that no less than 44 were definitely positive and 36 doubtful; out of 75 from San Thomé Island 4 were positive.

In an interesting discussion of these results the author points out that no cases of yellow fever have been recorded from Angola since the epidemic of 1860 to 1870; and never from San Thomé. Attention is called to similar puzzling results of these tests in other parts of Africa, to which BOYÉ has drawn attention [see above]. It is aptly pointed out that no biological test of this nature is infallible, and the possibility of antibodies being developed in the absence of the specific infections is well shown in the case of placental blood which contains antibodies against measles, diphtheria, poliomyelitis, and scarlet fever. These incertitudes indicate that one should hesitate before accepting the view that yellow fever may exist in two forms, one producing typical clinical symptoms and the other no changes except the development of protective antibodies. The author prefers to adhere to the experience of more than 30 years which shows that by the application of ordinary prophylactic measures against yellow fever—by which is meant actual clinically recognizable cases of the disease—successive epidemics have been brought under control, and he refuses to have any exaggerated fears of the " vague phantom " created from the results of serum protection tests. *E. H.*

JAMES (S. P.). Renseignements sur la fièvre jaune reçus pendant les six mois se terminant au 31 mars 1934. [**Information on Yellow Fever received during the Six Months ending 31st March, 1934.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. June. Vol. 26. No. 6. pp. 1048–1056. With 1 folding map.

The results of applying the mouse protection test for the examination of the sera of natives of the Anglo-Egyptian Sudan are shown in the following table, which includes figures obtained up to the end of December, 1933.

Province.	Locality.	No. of sera tested.	No. of positive results.
Blue Nile	Khartoum	36	1
,,	Wadi Medani	3	0
White Nile	Kosti	26	4
Upper Nile	Malakal	50	1
Mongalla	Juba	55	6
Bahr-el-Ghazal	Wau	27	7
,, ,,	Rumbek	30	17

The four positive results from Kosti and the one from Khartoum were obtained with the sera of non-residents of these localities.

These very important results are discussed by Dr. W. A. SAWYER in a letter to the author. The existence of such a high proportion of sera giving protection against yellow fever virus is considered to be a clear proof that they have been infected, but gives no information as to what proportion of the immune population may have shown any characteristic clinical symptoms, since during an epidemic the number of persons immunized is always much higher than the number of cases diagnosed clinically. It is very difficult to explain these " silent zones " of yellow fever in which an appreciable proportion of the population shows immunity, although no cases of clinical yellow fever have yet been observed. Possibly there are different strains, for a Brazilian strain, F. W., studied both in Brazil and New York, generally produces only fever in monkeys, without any other obvious symptoms. Yet in two cases this strain has produced typical fatal yellow fever with the same lesions as the most virulent strains.

Dr. G. M. FINDLAY also summarizes reasons for considering the mouse protection tests as specific for yellow fever.

Dr. G. B. SYMES gives particulars of the examination for mosquitoes of 30 aeroplanes at Kisumu and Nairobi, between June and December, 1933. In 18 aeroplanes *Anopheles* were found, and in 12, *Culex* ; also tabanids were frequently present. The insects were found in the passenger's cabin or the small compartment between this and the pilot's cabin.

In addition the article contains summaries of other observations on yellow fever which are summarized under the names of their respective authors. *E. H.*

HEWER (T. F.). **Yellow Fever in the Anglo-Egyptian Sudan. Serological and Post-Mortem Evidence.**—*Lancet.* 1934. Sept. 1. pp. 496–499. With 1 map.

During the last two years sera have been collected from representative groups of adults and children in the southern and western provinces of the Sudan and in the central district of Khartoum. These have been examined at the yellow fever laboratories of the Rockefeller Foundation at New York, by means of the mouse protection test and the results are of very great interest, for a wide zone has been discovered in which the disease appears to be, or to have been, endemic.

At Wau, 7 out of 27, and at Rumbek, 17 out of 30, adults, had positive sera. At Juba, out of 28 adults 3 were positive, and 3 gave partial protection, and 3 out of 22 children were also positive. At Malakal, another Imperial Airways port, among 55 persons one was positive and 3 inconclusive. Summarizing the results it would seem that yellow fever was present probably in endemic form in the Bahr-el-Ghazal six years ago ; in Mongalla seven years ago ; in the Upper Nile Province between eleven and eighteen years ago ; and in the Nuba mountains thirty years ago. There is no evidence of the disease ever having been present in the North of the Sudan, all sera from Khartoum and El Obeid being negative. In addition a fatal case of the disease with typical clinical symptoms occurred in a native of Wau during June 1934, and the diagnosis was confirmed by histological examination of the liver which showed advanced necrotic changes and Councilman lesions.

An interesting corroboration of the previous existence of yellow fever in Darfur is furnished in a recent article by Prince Omar Tousson, who mentions that during the Mexican War in 1863, 453 Sudanese soldiers from Darfur and Kordofan were sent to Vera Cruz where the mortality from yellow fever among Europeans was very high. African troops were in great demand because they were known to be immune to yellow fever and of these Sudanese only one, their commanding officer, died of the disease.

The difficulty of diagnosing mild or even fatal attacks of yellow fever is well known, and in a discussion of the apparent absence of any case of the disease among the foreign residents the author points out that in an area of 293,000 square miles, there are only about 700 persons who are at risk and in whose case a clinical diagnosis can be made. The possible danger of the spread of the disease by air and measures adopted for preventing it are described in detail and follow the general lines laid down in the International Convention for Aerial Navigation, to which the Sudan Government has become a signatory. *E. H.*

Boyé. Répartition des cas de fièvre jaune signalés en Afrique Occidentale au cours de l'année 1933. [**Distribution of Cases of Yellow Fever reported from West Africa during 1933.**]—*Bull. Office. Internat. d'Hyg. Publique.* 1934. June. Vol. 26. No. 6. pp. 1045–1047. With 1 map.

During 1933, in addition to the epidemic in the Bissagos Islands, cases have been recorded from the following provinces: Senegal, Niger Colony, French Guinea, Ivory Coast, Togoland, Gold Coast and Nigeria. These occurred in Europeans, 29 cases with 27 deaths; natives, 13 cases with 9 deaths; and Syrians, 7 cases with 6 deaths. The wide distribution of these cases is noteworthy and clearly indicates the necessity of continued sanitary supervision to prevent the development of epidemics. *E. H.*

Soper (Fred L.). **Some Notes on the Epidemiology of Yellow Fever in Brasil.**—Reprinted from *Rev. Hyg. e Saude Publica.* 1934. Feb. & Mar. Vol. 8. Nos. 2 & 3. pp. 37–61; 73–94. With 21 maps (1 folding), 1 chart & 3 figs.

These interesting notes on the epidemiology of yellow fever in Brazil are based on the observations of the Co-operative Yellow Fever Service maintained by the National Department of Health of Brazil and the Rockefeller Foundation.

Details are given of the epidemic at Cambuci, a small town of 823 inhabitants in the State of Rio de Janeiro, which has been carefully studied since the outbreak of yellow fever in 1928. The results of laboratory tests of 555 samples of sera show that approximately 60 per cent. of the population were immune, but the number of clinically recognized cases of the disease was only 13, with 5 deaths, between November 1930 and February 1931. It seems evident that the disease was widespread in a mild form, and the examination of the sera of persons " with a history of recent illness " compared with those without such a history supports this view, for a high degree of association was found between positive history of recent illness and positive protection test result (89·5 per cent. as compared with 46·8 per cent. in those without history of recent illness).

The symptoms among individuals who reported recent illness at this time are collected in the following table :—

Distribution of declared symptoms and signs among individuals reporting recent illness by age-groups.

Age group	Census.	History of illness Oct., 1930, Mar., 1931.	Percentage of group with history of illness.	Fever.	Headache.	Body Pains.	In Bed.	Nausea.	Vomiting.	Icterus.	Epigastric pain.	Conjunctival Congestion.	Ulorrhagia.
0–4	89	17	19·1±2·8	17	16	15	15	11	13	2	1	0	0
5–9	105	33	31·4±3·1	32	29	27	28	13	14	3	4	0	1
10–14	94	26	27·7±3·1	25	25	23	24	8	8	1	2	0	0
15–19	121	41	33·9±2·9	40	40	40	34	11	11	4	3	2	0
20–29	157	49	31·2±2·5	48	49	48	48	20	18	6	5	2	0
30–39	98	25	25·5±3·0	25	24	23	20	11	8	0	2	1	0
40–49	79	20	25·3±3·3	20	20	20	17	10	8	6	4	0	1
50–59	45	11	24·4±4·3	11	11	11	11	2	3	0	0	0	0
60–69	17	4	23·5±6·9	4	4	4	4	1	0	0	0	0	0
+70	18	1	5·6±3·7	1	1	1	1	0	0	0	0	0	0
Total	823	227	27·6±1·0	223	219	212	202	87	83	22	21	5	2
Percentage of persons ill reporting a given symptom				98·2	96·5	93·4	89·0	38·3	36·6	9·7	9·3	2·2	0·9

Additional notes are given on the rural epidemic of yellow fever, without *Aëdes aegypti*, in the Valle do Chanaan, Espirito Santo, Brazil, in 1932, and also on the value of the viscerotome for the removal of liver tissue from fatal cases for the discovery of silent yellow fever foci [*ante*, p. 77 and below].

In conclusion the author discusses the organization of the Yellow Fever Service in Brazil and calls attention to the urgent necessity of the disease being considered as a continental problem in South America, in view of the widespread distribution of the mosquito and the increased means of rapid transportation. This continental program of work should comprise :—

" (1) Anti-larval services in all principal cities and in all ports in Tropical America. This measure should prevent the future widespread dissemination of virus, and should greatly reduce the possibility of its international spread.

" (2) Protection test surveys to outline the recent distribution of yellow fever. This will undoubtedly be found much greater than is now believed.

" (3) Routine collection and examination of liver specimens from rapidly fatal febrile cases from all parts of possibly endemic areas. Smaller towns and rural areas are especially important.

" (4) Careful study of all places shown to be infected by the examination of liver tissue, with special reference to the possibility of vectors other than *Aëdes aegypti* and of vertebrate hosts other than man.

" (5) Anti-larval services in all towns and villages in and about known infected areas." *E. H.*

SOPER (Fred L.), RICKARD (E. R.) & CRAWFORD (P. J.). **The Routine Post-Mortem Removal of Liver Tissue from Rapidly Fatal Fever Cases for the Discovery of Silent Yellow Fever Foci.**—*Amer. Jl. Hyg.* 1934. May. Vol. 19. No. 3. pp. 549–566. With 3 figs. & 2 maps. [12 refs.]

The desirability of obtaining samples of liver from rapidly fatal febrile cases in areas liable to infection with yellow fever has led the authors to devise an instrument—the viscerotome—which can be used by laymen without autopsy for the rapid removal of liver tissue. The construction of this instrument is shown in the accompanying figures and its use has made possible the collection of liver specimens by non-medical personnel, over a wide area, with a minimum of expense and trouble. General regulations for its use in Brazil were included in a Presidential Decree of May 23, 1932, and the results of examinations are of very great interest. Between May, 1930 and June 1933, 28,468 tissues were examined with 75 positive diagnoses of yellow fever, of which only 21 had been diagnosed clinically as suspected cases of the disease. With the exception of one case from Bolivia all these came from various parts of Brazil, mainly from its north-east corner. Typical fatal cases of the disease were found in otherwise silent endemic foci,

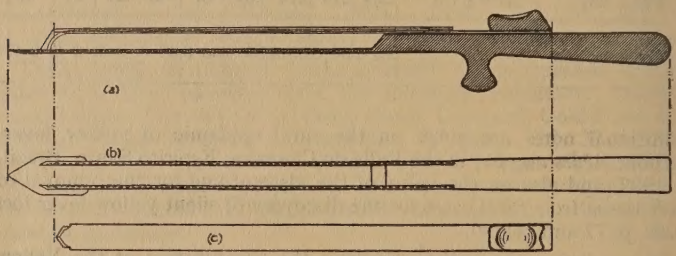

Fig. 1. (*a*) Sectional view of viscerotome with sliding blade in cutting position. (*b*) Plane view of viscerotome with sliding blade withdrawn. (*c*) Sliding blade.

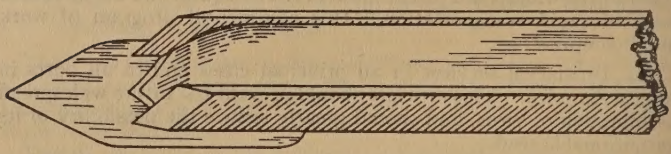

Fig. 2. Distal portion of viscerotome showing position of sliding blade during introduction and withdrawal from the body.

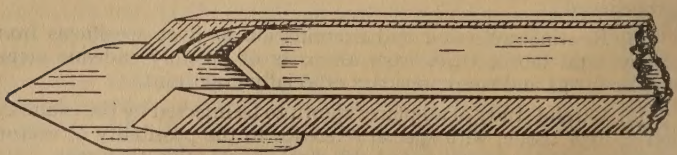

Fig. 3. Distal portion of viscerotome showing position of sliding blade during cutting of liver tissue.

[Reproduced from the *American Journal of Hygiene.*]

and in one village, the presence of yellow fever without Aëdes, was confirmed 11 months after the occurrence of the first suspect case. The results, however, in general confirm the absence of yellow fever virus from key-centres of population and also from hundreds of towns and villages in which anti-mosquito measures are being applied. In addition valuable data have been accumulated regarding the distribution of other diseases producing characteristic liver lesions. *E. H.*

Sawyer (W. A.). L'enquête sur l'immunité vis-à-vis de la fièvre jaune, au moyen de l'épreuve de protection de la souris. [**The Study of Immunity against Yellow Fever by Means of the Mouse Protection Test.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. June. Vol. 26. No. 6. pp. 1057–1060. With 1 folding map.

A brief summary of the work undertaken by the International Health Division of the Rockefeller Foundation on the distribution of yellow fever, by the examination of specimens of sera collected from various localities, and tested in New York, Lagos or Bahia, generally by means of the mouse protection test.

Approximately 25,000 sera have been examined up to date, and the only modification of the original method of using the test [see this *Bulletin*, Vol. 29, p. 198] has been to increase the strength of the infected mouse brain suspension from 10 to 20 per cent. The results of the examination of several hundred sera from non-endemic areas shows that less than 1 per cent. have given a positive reaction, these cases doubtless being due to various causes of error. The specificity of the test is clearly indicated by the great number of positive results obtained in many endemic areas. The results support the view that yellow fever immunity is confined to two great regions, one in South America and the other in Africa ranging through West and Central Africa to the western frontier of the Anglo-Egyptian Sudan. All recent epidemics have occurred in these areas, but the regions in which immunity is found are very much more extensive than one would have expected from previous knowledge of the disease. *E. H.*

Sawyer (W. A.). Notre expérience de la vaccination contre la fièvre jaune. [**Our Experience with Vaccination against Yellow Fever.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. June. Vol. 26. No. 6. pp. 1072–1074.

A total of 56 cases have been vaccinated in the laboratories of the Rockefeller Foundation by the use of human immune serum and neurotropic mouse virus, following the method recommended by Sawyer, Kitchen & Lloyd [see this *Bulletin*, Vol. 29, p. 572]. The results indicate that this method is very efficacious and the sera of 35 persons tested before and after vaccination showed in every case the development of protective properties.

The sera of 11 persons collected 2 years after vaccination showed a slight diminution in the titre, and possibly it may be necessary to examine persons exposed to infection after an interval of 3 years and when the titre is very low, revaccinate the patient. Since the introduction of this method of vaccination no case of the disease has occurred among the yellow fever personnel of the International Health Division, whilst previously accidental infections seemed to be unavoidable.

E. H.

PETTIT (Auguste) & STEFANOPOULO (Georges J.). La vaccination antiamarile à l'Institut Pasteur. [**Vaccination against Yellow Fever at the Pasteur Institute.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. June. Vol. 26. No. 6. pp. 1075–1077.

The authors have vaccinated 12 subjects aged 23 to 48 years by the serum-virus method, in 10 cases using horse anti-yellow fever serum, and in the other two human convalescent serum. The results were quite satisfactory and show that horse anti-serum can replace human convalescent serum. *E. H.*

LAIGRET (Jean). Sur la vaccination contre la fièvre jaune par le virus de Max Theiler. [**Vaccination against Yellow Fever with Max Theiler's Virus.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. June. Vol. 26. No. 6. pp. 1078–1082.

The author advocates the use of three injections of neurotropic virus without any immune serum, as it has been found that the inoculation of small doses of this virus fixed for mice is followed by the development of immunity without any serious general reactions [see this *Bulletin*, Vol. 29, p. 572]. *E. H.*

ADVIER (M.). L'épreuve de Max Theiler (séro-protection de la souris blanche contre le virus amaril neurotrope) pourrait-elle être remplacée par une intradermo-réaction ? [**Can Max Theiler's Test (the Serum Protection of White Mice against Neurotropic Yellow Fever Virus) be replaced by an Intradermal Reaction ?**]—*Bull. Soc. Path. Exot.* 1934. June 13. Vol. 27. No. 6. pp. 576–578.

The author finds that the intradermal inoculation of 0·1 cc. of a 1 per cent. suspension in saline of infected mouse brain is followed by the development of a dermal reaction in the case of patients immune against yellow fever.

Eighteen patients were tested, of whom 5 gave a positive reaction ; this appeared about 24 hours after the inoculation, attained its maximum intensity on the second day, and gradually disappeared on the fourth day. The other 13 patients remained negative. The test was repeated 15 days later with the positive cases and gave the same results.

The blood of these positive cases and three negative ones was tested in mice but gave inconclusive results. Nevertheless 4 out of the 5 cases giving the positive intradermal reaction had been previously vaccinated against yellow fever by the inoculation of neurotropic virus. Three of those giving a negative reaction were then vaccinated by the same method and when tested 15 days later, two of them gave a positive dermal reaction. It would seem, therefore, that this reaction offers distinct possibilities. *E. H.*

LLOYD (Wray) & MAHAFFY (Alexander F.). **The Serum Antibody Titer of *Macacus rhesus* following Repeated Inoculations of Yellow Fever Virus.**—*Jl. Immunology.* 1934. Apr. Vol. 26. No. 4. pp. 313–320.

The author's results show that no significant alteration took place in the protecting antibody titres of the sera of five rhesus monkeys, immune to yellow fever, after repeated subcutaneous injections of neurotropic yellow fever virus at monthly intervals.

Three of these monkeys had originally been inoculated with neurotropic virus from either mouse or monkey, whilst the other two had been exposed to infection with the viscerotropic virus, both by the bites of infected mosquitoes and infected blood. The sera of these five monkeys were tested for their antibody titre against yellow fever virus by means of intraperitoneal protection tests, using six mice for each test. After the initial titration each immune monkey was tested at monthly intervals for three consecutive months, after receiving an injection of neurotropic mouse virus, and the results clearly indicate that there was no appreciable alteration in the antibody content.

E. H.

Davis (Nelson C.). **On the Use of Immune Serum at Various Intervals after the Inoculation of Yellow Fever Virus into Rhesus Monkeys.**—*Jl. Immunology.* 1934. May. Vol. 26. No. 5. pp. 361–390.

The effects of immune serum from recovered animals have been tested in a long series of monkeys, and it was found that when injected 24 to 48 hours following the inoculation of virus, the serum was capable of preventing fever or ameliorating the disease in a significant proportion of experimental monkeys.

One of the great difficulties in apportioning the effects of the treatment was the variation in the resistance of the animals themselves. One monkey died with typical yellow fever lesions only 50 hours after the injection of virus, whilst at least a dozen monkeys in the laboratory had incubation periods of 3 weeks or more.

The author's results, given in detail in tabular form, show that 2 to 3 cc. of serum per kilo body weight, given 24 hours after injection of the virus, would save more than half the animals ; and if the dosage of virus was small 5 cc. of serum would save at least half of them when given after an interval of 48 hours. Once the disease had definitely developed, as shown by a temperature of 104°F. or more, even fantastically large amounts of serum had no effect.

In discussing the use of immune serum for therapeutic purposes in human beings, the difference in the course of the disease in the two hosts, man and monkey, is emphasized. Finally, a case is described of a laboratory worker who seems to have shown a favourable reaction as a result of the combined effects of prophylactic and therapeutic doses, after being accidentally exposed to infection with yellow fever virus.

E. H.

Mathis (M.). Vaccination antiamarile du singe, à l'aide de virus vivant provenant du cobaye mort de la maladie expérimentale. [**Yellow Fever Vaccination of the Monkey, by Liver Virus obtained from Guineapigs dying from Experimental Infection.**]—*Bull. Soc. Path. Exot.* 1934. June 13. Vol. 27. No. 6. pp. 505–510. [12 refs.]

A rhesus monkey was inoculated both intraperitoneally and subcutaneously with a total dose of 2 cc. of a 5 per cent. suspension of the brain of a guineapig, infected with a strain of yellow fever that had undergone 155 passages in mice and more than 10 passages in guineapigs. The monkey showed no signs of disease but from the 2nd to the 5th day virus pathogenic to mice was recovered from its blood. This

animal was repeatedly exposed to infection with yellow fever but was resistant. Three other monkeys also vaccinated in different ways with the virus from guineapigs gave similar results. *E. H.*

Hoskins (Meredith). **Protective Properties against Yellow Fever Virus in the Sera of the Offspring of Immune Rhesus Monkeys.**—*Jl. Immunology.* 1934. May. Vol. 26. No. 5. pp. 391–394.

Tests were made with the sera of 5 baby monkeys born of immune mothers. Four of the mothers had been infected with the African (Asibi) strain and one with a Brazilian (S. R.) strain of yellow fever virus before the period of gestation. All the sera were tested when the babies were 6 months old and still feeding upon the mother's milk, and two were bled again 3 months later after having been separated from the mother for 3 weeks.

The results of protection tests in mice show that the sera of all 5 baby monkeys contained protective bodies against yellow fever at the age of 6 months. This protective ability had completely disappeared in the two examined three months later and in addition the sera of two other monkeys respectively 13 and 23 months old, born of immune parents, failed to protect. Subsequently the author has examined the sera of other baby monkeys born to mothers immune to yellow fever. Within 12 hours of birth the sera of these babies was found to contain a relatively high titre of immune bodies, which suggests that the protective bodies may be derived from the mother through the placenta, rather than through the maternal milk.

These interesting observations may be of significance from an epidemiological standpoint as positive protection tests obtained from relatively young children may be the result of an immunity transmitted from the parent to offspring and not the result of actual infection.

E. H.

Snijders (E. P.), Postmus (S.) & Schüffner (W. A. P.). Over de beschermende werking van gele-koorts- en denguesera tegenover gele-koortsvirus. [**On the Protective Action of Yellow Fever and Dengue Sera against Yellow Fever Virus.**]—*Nederl. Tijdschr. v. Geneesk.* 1934. July 14. Vol. 78. No. 28. pp. 3253–3272. [Refs. in footnotes.] English summary.

The authors discuss the possibility that under certain conditions dengue might provide some grade of immunity against yellow fever, but hitherto the results of all investigations have failed to show the presence of any humoral immunity.

A modification of the mouse protection test was used for the repeated examination of the sera of 20 cases of experimental Sumatran and Javanese dengue, but no evidence was ever obtained of any protective action against yellow fever virus. The examination of the sera of 5 persons working regularly in the yellow fever laboratory showed the existence of weak protective action in two of them, which is believed to be the result of slight (abortive) infection. A comparison of the two diseases shows that in the case of dengue immunity varies greatly in

different individuals and antibodies are not found in the serum, whereas in yellow fever a practically absolute immunity always develops, with abundant antibodies in the serum. *E. H.*

Davis (Nelson C.). **Attempts to determine the Amount of Yellow Fever Virus injected by the Bite of a Single Infected Stegomyia Mosquito.** —*Amer. Jl. Trop. Med.* 1934. July. Vol. 14. No. 4. pp. 343–354.

Aëdes aegypti infected with yellow fever virus were allowed to feed upon new-born white mice, and the latter were immediately killed and ground up with powdered glass and monkey serum. The resulting paste was mixed with 15 per cent. monkey serum and the liquid removed by centrifuging, this process being repeated four times in order to extract any virus from the tissue. Varying dilutions of the extract were then inoculated into a series of rhesus monkeys. In two experiments the titrations indicated that each mosquito had injected at least 100 infective doses of virus.

Mosquitoes from one lot, which was later used in a feeding experiment on a mouse, were titrated immediately following the infective blood meal and again after ten days. There was a decrease in titre during this interval, which confirms the view that yellow fever virus within the mosquito seems not to increase but rather to diminish in quantity [see this *Bulletin*, Vol. 31, p. 81].

From a comparison of the amounts of virus in whole insects and in mice bitten by mosquitoes of the same lot, it would seem that about 1 per cent. of the total virus content was injected at each feed. *E. H.*

Findlay (G. M.) & Clarke (L. P.). **The Susceptibility of the Hedgehog to Yellow Fever. I.—The Viscerotropic Virus.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1934. Aug. 4. Vol. 28. No. 2. pp. 193–200. With 6 figs. on 1 plate. [20 refs.]

The authors give details of their interesting observation that the common hedgehog, *Erinaceus europaeus*, is very susceptible to infection with the viscerotropic strain of yellow fever virus.

Twenty-eight animals were infected by the subcutaneous or intraperitoneal inoculation of material from either monkeys or hedgehogs dying of yellow fever, and death invariably occurred in from 4 to 7 days. The naked eye and histological changes were very similar to those met with in man and rhesus monkeys dying of yellow fever, and virus was present at death in the blood, liver and kidneys.

Repeated passages were carried out in hedgehogs with material filtered through Berkefeld filters, and after seven passages the liver filtrate was still virulent for rhesus monkeys. Monkeys immune to known strains of yellow fever virus were also immune to the hedgehog passage strain.

Filtrates of hedgehog livers inoculated intracerebrally into mice produced encephalitis histologically similar to that caused by neurotropic strains of yellow fever virus. The mice died with encephalitic symptoms in from 8 to 18 days, but in subsequent passages in mice the incubation period was gradually reduced.

In view of the wide distribution of the Erinaceidae in Africa it is possible that the hedgehog, and perhaps other Insectivora, may serve as a virus reservoir for the maintenance of infected mosquitoes. *E. H.*

Stefanopoulo (G. J.). Recherches sur la fièvre jaune expérimentale de la souris et du cobaye. [**Studies on Experimental Yellow Fever in Mice and Guineapigs.**]—*Ann. Inst. Pasteur.* 1934. May. Vol. 52. No. 5. pp. 553–595. With 7 figs. & 4 diagrams. [38 refs.]

This memoir is a valuable summary of the author's work on the subject and his results are discussed in relation with the results obtained by other workers. Concerning the susceptibility of white mice to the neurotropic virus, the author found that strains of these animals from different localities, including France, Holland and Belgium were equally susceptible. The common grey house-mouse was also found to be susceptible and a strain of yellow fever was maintained exclusively in grey mice for 20 passages in about seven months, without the virus showing any difference in its effects from those obtained in white mice.

When mice were inoculated intraperitoneally the virus was found in the brain, even on the first day, in greater quantities than in the circulating blood, but the blood of any mice that survived either intraperitoneal or intracerebral injections always contained antibodies against the infection.

Guineapigs were inoculated intracerebrally with various strains of the virus and found to be very susceptible to virus which had been through a certain number of passages in mice. They generally died of myelo-encephalitis about 8 or 9 days after inoculation, and many of them showed more or less pronounced fatty degeneration of the liver.

The author's results confirm the view that there are differences in behaviour between the viscerotropic and neurotropic viruses, the degree of neurotropism in any particular strain varying according to the number of passages in mice which it has undergone. In using any such strain for the immunization of human subjects, it is necessary to consider the origin, age, and method of preservation of the virus, in the choice of the procedure which should be applied. *E. H.*

Nicolau (S.), Kopciowska (L.) & Mathis (M.). Inclusions produites par le virus de la fièvre jaune chez les souris inoculées sous la dure-mère. [**Inclusions produced by Yellow Fever Virus in Mice inoculated beneath the Dura Mater.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 24. pp. 818–820.

——, ——, —— & Baffé (O.). Les inclusions intranucléaires chez le cobaye infecté par voie sous-dure-mérienne avec le virus de la fièvre jaune. [**Intranuclear Inclusions in Guineapigs infected Subdurally.**]—*Ibid.* 820–22.

The authors have studied the brains of 55 mice infected with yellow fever virus, 32 inoculated with the ordinary viscerotropic strain from monkeys, and 23 with a neurotropic strain from mice. All the mice dying as a result of the infection showed inclusions in the nerve cells of the brain and spinal cord. The inclusions were also found in the glial cells, the endothelial cells of the small brain capillaries, the cells of the choroid plexus, the lining of the walls of the lateral ventricles and Purkinje's cells. In Ammon's horn the acidophile bodies reached a diameter of 2–4μ, but even in these large bodies no internal structure could be detected. The bodies were also easily found in the nuclei of the thalamus, and in the anterior horns of the spinal cord nearest the brain.

The pathological lesions in the central nervous system consist of an encephalo-myelitis, sometimes with destruction of the anterior

horns of the spinal cord, as in Borna disease. By killing mice at daily intervals after inoculation, all stages were obtained in the transformation of the karyoplasm of the nerve cells into yellow fever inclusion bodies. The first bodies appeared on the 4th day in the nerve cells of the brain, and a little later in some of the glial cells. In animals dying 11 to 14 days after inoculation, there was a certain absorption of the brain lesions with a partial and progressive disappearance of the inclusions, first in the brain and then in the spinal cord. The last cells in such cases to show acidophil inclusions are the glial cells.

It is important not to confuse the oxychromatic degeneration of the nucleus, caused by its death, with the presence of inclusions resulting from the resistance of the cell. Inclusion bodies were never found in the hepatic cells of the infected mice.

The virus studied, which had been passaged in the brains of mice for several generations, was found to be not strictly neurotropic, and, contrary to the statements of certain authors, did not produce " septinevritis " in the nervous system of experimental animals.

The second article is concerned with the examination of guineapigs infected by intracerebral inoculation of the virus. The resulting histological changes were almost identical with those recorded above in the case of mice, but it is of interest that inclusion bodies were found in the central nervous system of guineapigs showing no other pathological changes. The characteristic yellow fever bodies are considered to be the oxyphilic spherules occurring singly or in groups, sometimes surrounded by a halo, but always surrounded by karyoplasm which preserves the staining properties. In the nerve cells the inclusions were nearly always multiple (8–10 or even 20) ; but in the glial cells there were rarely more than 6 inclusions and often only a single large body 2–3μ in diameter. The cells of the choroid plexus generally showed only single bodies in the nucleus, surrounded by a large halo. The livers of guineapigs dying of this infection showed very much reduced lesions of the liver compared with those in monkeys, and intranuclear inclusions were only found very exceptionally. *E. H.*

i. Nicolau (S.), Kopciowska (L.), Balmus (G.) & Mathis (M.). Inclusions provoquées par le virus de la fièvre jaune chez le singe. [**Inclusions produced by Yellow Fever Virus in the Monkey.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 25. pp. 944–947.

ii.——, —— & Mathis (M.). Sur les inclusions amariles ; leur rapport avec les inclusions provoquées par d'autres ultravirus. [**Their Relation to the Inclusions produced by other Virus Infections.**]—*Ibid.* pp. 947–950.

i. As a result of the examination of 14 *Macacus rhesus* infected in various ways with yellow fever virus (intracerebrally, intraperitoneally and by bites of mosquitoes) the authors have arrived at the following conclusions :—

With all three methods of infection the alterations in the nervous system are identical but very slight, consisting of a more or less distinct proliferation of the glial cells of the brain and spinal cord and other changes in the nervous tissue itself. The inclusion bodies are sometimes present in the nuclei of the nerve cells of the brain, also in Purkinje's cells, and exceptionally in certain cells of the spinal cord. The glial cells contain inclusion bodies more often than the ganglion cells. When stained with Mann, the inclusions frequently appear blue,

surrounded by a halo without the characteristic oxyphilic granules. In all the monkeys examined (10) the nerve cells of the spinal ganglia contained typical intranuclear inclusion bodies, which were found also in certain intracardiac nerve cells and in the walls of the intestine. They were absent from the cells of the parotid, pancreas, cardiac muscle, lymphatic ganglion, lung, suprarenal and kidney. In the spleen, in addition to the lesions described by previous authors, very well developed inclusion bodies were often found in the large endothelial cells. The histological changes were identical in monkeys dying 5 or 6 days after inoculation, and those dead after 15 days. The livers of all the monkeys showed the characteristic changes and inclusion bodies.

ii. This article is a further discussion of the interpretation of the nuclear changes observed in certain cells of animals infected with yellow fever virus. The virus is assumed to enter the nerve cell and become centred in the nucleus which as a result undergoes various changes. The nucleoplasm first becomes granular, then particles of chromatin appear among the granules. These chromatic bodies become rounded off and surrounded by a halo and either single or multiple oxyphilic centres appear in these bodies. These oxyphilic changes gradually extend until the bodies are entirely acidophile, surrounded by the remains of the nucleus which preserves its original staining qualities. In addition to these changes some of the nuclei of cells which presumably have succumbed to the action of the virus undergo oxyphilic degeneration of the nucleus, involving the whole of the chromatin, which forms irregular masses of degenerated material sometimes arranged around the periphery of the nucleus. The authors found that this type of degeneration could be produced in the livers of rabbits by the inoculation of tuberculin or vibrion septique toxin.

Finally, as previously noted by Cowdry and Kitchen [*Science*, Vol. 69, p. 252] it is considered that there is a close resemblance between the yellow fever inclusion bodies of the nerve cells and the Joest-Degen bodies found in Borna disease. *E. H.*

Nicolau (S.) Kopciowska (L.) & Mathis (M.). Inclusions amariles chez des animaux réputés réfractaires à l'action pathogène du virus de la fièvre jaune. [**Yellow Fever Inclusion Bodies in Animals considered Refractory.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 27. pp. 1332–1334.

Rabbits, including both young and adults, were inoculated intracerebrally, intraperitoneally or into the sciatic nerve with either viscerotropic yellow fever virus from monkeys, or neurotropic virus from mice. Inclusion bodies characteristic of yellow fever were found in the nerve cells and glial cells of the central nervous system of many of the 22 animals examined, but these bodies were not so numerous nor so well developed as those in mice and guineapigs. The bodies were never found in the liver cells of inoculated rabbits. *E. H.*

Haagen (Eugen). **Yellow Fever Virus in Tissue Culture.**—Reprinted from *Arch. f. Experim. Zellforschung.* 1934. Vol. 15. pp. 405–421. With 8 figs.

This article contains a useful summary of the author's results of studying yellow fever virus in tissue culture including full details of

the methods employed [see this *Bulletin*, Vol. 29, p. 577 and Vol. 30, p. 357].

The most satisfactory medium was found to be normal monkey serum diluted ten times with tyrode solution containing living fowl-embryo cells. Living cells are essential for growth. The histological examination of rabbit tissues infected *in vitro* with yellow fever virus showed intranuclear changes in the affected cells which were most distinct in the corneal and testicular tissues. The nuclear chromatin showed acidophile granulation similar to the changes in the tissues of infected animals. *E. H.*

Mathis (Maurice). Agressivité et pontes comparées du moustique de la fièvre jaune en conditions expérimentales. [**Comparison of the Aggressiveness and Egg-laying of** *A. aegypti* **under Experimental Conditions.**]—*C. R. Soc. Biol.* 1934. Vol. 115. No. 14. pp. 1624–1626.

Using *Aëdes aegypti* bred under experimental conditions, it was found that when fed on man the adult females only exceptionally required a second meal of blood before laying eggs. On the other hand, those fed on monkeys, rabbits, or guineapigs, generally required two meals before laying, a fact which suggests that man is the most favourable host. In addition, the number of eggs per female laid by mosquitoes fed on various hosts was greater when human blood had been ingested as shown by the following table :—

	Number of eggs per female fed on			
	Man.	Monkey.	Rabbit.	Guineapig.
First egg laying	46	34	38	35
Second egg laying ...	50	42	33	35
Total	96	76	71	70

E. H.

Mathis (Maurice) & Baffet (Odette). Développement larvaire du moustique de la fièvre jaune en culture pure d'Euglènes. [**Development of Larvae of** *A. aegypti* **in Pure Culture of Euglena.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 19. pp. 317–319.

The authors obtained eggs of *Aëdes aegypti* which were alive but bacteriologically sterile. They allowed them to hatch in several media, testing for sterility from time to time. In peptone water the larvae died in a few days, none having passed beyond the second stage. When a fragment of brain was added to the peptone water, no better results were obtained [but it is not clear whether the brain was autoclaved in the medium or added aseptically to the sterile peptone water]. Living *Euglena* were available in pure culture. In this the larvae hatched and fed well, becoming adults very quickly. Tests for bacteriological sterility were made, but the technique is not mentioned : [it is not clear that the workers excluded anaerobes or organisms which live at room temperature better than at 37°C]. *P. A. Buxton.*

Taylor (A. W.). **A Note on Mosquitos breeding in Tree-Holes in Northern Nigeria.**—*Bull. Entom. Res.* 1934. July. Vol. 25. Pt. 2. pp. 191–193.

Although the climate of Northern Nigeria is very different from that of the Southern Provinces, a true tree-hole mosquito fauna nevertheless exists at least as far north as Gadau (lat. 12°N.). Here, near the European station, 11 holes were discovered in as many trees belonging to 7 species. From the beginning of July, 1931, until the second week of September the holes contained water, and the larvae found on periodic visits were bred out in the laboratory. No preference on the part of the mosquitoes for particular species of trees was observed.

Like the climate itself, the tree-hole mosquito fauna of N. Nigeria differs widely from that of the South. *Aëdes metallicus*, *A. furcifer* and *A. unilineatus*, which are absent, and *A. simpsoni*, which is rare, in the South, abound in tree-holes in the North. *Aëdes africanus*, *A. stokesi*, *A. apicoargenteus*, *A. wellmani* and *A. longipalpis*, all common in Southern tree-holes, were not met with during the present survey, in the course of which, in addition to those already mentioned, the following species were reared :—*Aëdes luteocephalus*, *A. aegypti*, *Culex nebulosus*, *C. decens* and *Megarhinus brevipalpis*.

The immersion in water of debris and scrapings, taken from the eleven tree-holes in January and February, resulted in the breeding out of *Aëdes simpsoni* from 10 holes, *A. unilineatus* from 5, *A. metallicus* from 2, and *A. aegypti* from a single hole, showing that in N. Nigeria the viability of tree-hole breeding Aëdes is preserved during the dry season. It is noted that, among the tree-hole breeders found, apart from *A. aegypti*, at least *A. luteocephalus* and *A. simpsoni* are experimentally capable of transmitting the virus of yellow fever.

E. E. Austen.

Haagen (E.). Das Gelbfieber. Neuere Forschungsergebnisse. [**Yellow Fever. Recent Investigations.**]—*Deut. Med. Woch.* 1934. June 29. Vol. 60. No. 26. pp. 983–988. [55 refs.]

A general summary of recent work on the subject. *E. H.*

Annales de Médecine et de Pharmacie Coloniales. 1934. Jan.–Feb.–Mar. Vol. 32. No. 1. pp. 78–84.—L'etat actuel de la vaccination contre la fièvre jaune. [**The Present Position of Vaccination against Yellow Fever.**]

A useful summary of the subject. *E. H.*

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

CUNNINGHAM (J.), THEODORE (J. H.) & FRASER (A. G. L.). **Further Observations on Indian Relapsing Fever. Part I. Types of Spirochaetes found in Experimental Infections.**—*Indian Jl. Med. Res.* 1934. July. Vol. 22. No. 1. pp. 105–155. With 25 charts & 1 fig. [29 refs.]

This article is a valuable contribution to the problem of the nature and origin of the various serological types of spirochaetes occurring in cases of Indian relapsing fever.

Cunningham previously showed that the spirochaete of the relapse differed serologically from the spirochaete present in the first attack, and that these two types alternated with each other [see this *Bulletin*, Vol. 23, p. 112]. In the present investigation these observations have been greatly extended and the study of experimental infections in squirrels (*Sciurus palmaris*) and monkeys, has resulted in the isolation of seven additional serological types, bringing the total up to nine. The majority of these new types were originally found in second relapses (third attacks) which occurred in the same animal in about 4 per cent. of the cases. These spirochaetal types were merely serological entities and showed no morphological differences. In the case of two types (H and I), however, differences were observed in the form of attack produced. The types are referred to by letters ; " A " is the original first attack type ; " B " the first relapse type ; " D," " E," " F," " G " and " H " were obtained from second relapses ; " C " also occurred in second relapses but was originally isolated from a mixed type infection ; " I " was found in a first relapse.

Types A, B, D and E were definite and stable, but the remainder were more liable to change. Type F was rarely found and did not persist for many subpassages, being generally supplanted by Type B.

A study of agglutinin curves following infections with the various types showed four forms following either the first attack or the relapse.

(*a*) A simple response to the type of spirochaete inoculated, or in the case of the relapse, to this type.

(*b*) A series of one or more subsidiary rises to other types following the main curve in succession, the summit of each succeeding curve being lower than its predecessor.

(*c*) One or more subsidiary curves to other types arising simultaneously with the main response to the type causing the infection (or the relapse).

(*d*) A combination of the two preceding forms (*b* and *c*) in the same infection or relapse.

Experiments to determine the origin of the various types gave the conception of an organism composed not so much of pre-existing antigenic elements as of potential variants. The change of type seems to be a gradual process and occurs at different times during the disease. Occasionally infections with more than one type of spirochaete have been observed.

A study of the spirochaetes in 506 first relapses shows that :—

(*a*) The appearance of any particular type in an infection invariably debars it from any further participation in the disease in the same

animal. (*b*) The primary types A and B are definitely complementary to each other. The remaining types occupy an intermediate position although C, D and E seem more closely related to B, and G to A; but there are no clearcut distinctions between them, nor can they be divided into associated groups.

An examination of a series of first relapses derived from three strains obtained from different human sources showed that the majority of them were due to types A and B. When the second relapse strains were followed through a succession of relapses it was found that there was a persistent tendency to revert to the primary types A and B.

Types H and I produced prolonged attacks (up to 19 and 20 days) and the spirochaetes seemed unable to develop in any numbers. The types either remained pure throughout the whole attack or changed to another type about the 11th day or later. The primary types A and B, and also D, E and G were found to be very stable on subpassage as long as the transfers were carried out 24 to 48 hours after infection.

Changes of type without the intervention of a relapse were observed on rare occasions. Types C, F and G were those most frequently involved.

The article is accompanied by numerous tables and charts illustrating the various points dealt with by the authors. [It should be read in its entirety by those interested in the subject.] *E. Hindle.*

Advier (M.), Alain (M.) & Riou (M.). Fréquence et aspects cliniques de la fièvre récurrente à spirochète de Dutton en Afrique Occidentale Française. [**Frequency and Clinical Aspects of Tick Fever** (*S. duttoni*) **in French West Africa.**]—*Bull. Soc. Path. Exot.* 1934. June 13. Vol. 27. No. 6. pp. 593–598.

During the course of two years the authors have observed 46 cases of tick fever among the patients of the hospitals and dispensaries. These patients comprised 13 Europeans and 33 natives, who with two exceptions had acquired the infection in Dakar or other parts of Senegal.

The difficulties in the diagnosis of this disease are well exemplified in these cases for only 25 of the patients showed spirochaetes in the blood and in these the parasites were usually extremely rare. The remaining 21 cases were diagnosed only by the inoculation of their blood into rats.

The clinical symptoms alone do not readily enable one to distinguish this disease from any other febrile infections. General indications comprise a sudden access of fever persisting for more than a few hours accompanied by a saburral state of the alimentary canal, nausea and vomiting, headache and muscular pains in the limbs. There is an absence of stupor or excitation, also of erythema or pharyngitis. Generally albuminuria is present but is only very slight and does not increase. The only complication, observed in 5 Europeans and 4 natives, was the result of invasion of the meninges, or of the central nervous system, by the spirochaete.

The disease is evidently widespread in French West Africa, but is generally benign, with a favourable prognosis. Nevertheless it requires very energetic therapeutic treatment to effect a cure, involving a course of injections of 914 or sulpharsenol, with increasing doses, as in the case of syphilis. *E. H.*

Mathis (C.) & Durieux (C.). Existence d'un foyer endémique de typhus récurrent, à Saint-Louis du Sénégal. [**The Existence of an Endemic Centre of Relapsing Fever, at St. Louis, Senegal.**]—*Bull. Acad. Méd.* 1934. June 19. 98th Year. 3rd Ser. Vol. 111. No. 23. pp. 839–844.

The authors have tested the infectivity of rodents collected in St. Louis and the neighbourhood by inoculating emulsions of their brains into white mice. Two lots each of 5 *Epimys decumanus*, collected in the south of the town, and one lot of 5 *Epimys golonda campanae* collected in the Sor district were found to be infected. Negative results were obtained with 2 lots respectively of 7 and 8 *Arvicanthus rufinus* captured outside the town, and a lot of 9 *Mastomys coucha* and 1 *A. rufinus*.

The strain isolated from the St. Louis rats was found to produce typical relapsing fever when inoculated into a human subject. After an incubation period of 5 days this patient had five attacks of 2 to 5 days each, alternating with apyretic intervals of 2 to 6 days in the course of a month, when the disease was cured by arsenical treatment.

Mice are susceptible to infection with this strain, but young rabbits, guineapigs and one monkey, *Cercopithecus patas*, all showed very slight infections.

The results of a cross-immunity experiment support the view that this strain is identical with the Dakar strain of *S. duttoni*, commonly referred to as *S. duttoni* var. *crocidurae*.

Finally, the authors discuss the nature of the spirochaete described under the name of *Spirillum pitheci* which was discovered by Thiroux and Dufougeré[1] in the blood of a *Cercopithecus patas* at St. Louis.

Its general description and the results of an experimental study of the strain by Laveran and Pettit[2] suggest that this spirochaete is probably also identical with the Dakar strain, which seems to have a wide distribution in French West Africa. *E. H.*

Coleman (George E.). **Relapsing Fever in California. III. The Carrier Condition ; Epidemiology. IV. Cross-Immunity : Susceptibility of Sierra Golden Mantle Ground Squirrels.**—*Jl. Infect. Dis.* 1934. May–June. Vol. 54. No. 3. pp. 281–294. [42 refs.] ; pp. 295–304. [11 refs.]

After a full discussion of the subject the author gives the results of experiments with the Californian strains of relapsing fever spirochaetes.

In mice, infection of the brain did not persist after the blood had ceased to be infective. Moreover, attempts to reactivate possible latent infections by the inoculation of vaccines into recovered mice also gave negative results. With reference to the epidemiology of the disease, the possibility of rodent reservoirs is supported by the observations of Porter, Beck and Stevens also in California [see this *Bulletin*, Vol. 30, p. 361], but in the district they visited the author, three months previously in March, failed to recover any spirochaetes from nine adult squirrels and a chipmunk, and also no ticks were found. The positive findings three months later were probably in young animals recently infected in their burrows by nymphal ticks.

[1] *C. R. Acad. Sci.* 1910. Vol. 150, pp. 132–135.

[2] *Bull. Soc. Path. Exot.* 1910, Vol. 3, pp. 419–422.

In the cross-immunity tests attention was paid to the degree and duration of the immunity and the relative virulence against each other of the two strains to be tested. A close relation was found to exist between various Californian strains even though some of these strains had their origin several hundred miles apart. Furthermore, serological evidence was obtained favouring the probability of a close relationship between the Texas and California strains.

Five Sierra golden-mantle ground squirrels, *Callospermophils chrysodeirus*, from Plumas County, were tested for spirochaetes in various ways—daily examinations of the blood, subinoculations into mice, agglutination, protection tests, etc.—but failed to give any evidence of infection. Finally, three were inoculated in various ways with three strains of the spirochaetes but failed to show any signs of infection. Moreover, their blood serum, both before and after the injection of these strains, failed to protect mice against infection with the same strains and also an additional one. It seems probable, therefore, that this species is immune against relapsing fever. *E. H.*

Varden (Arthur E.). **Relapsing Fever in Children.**—*Amer. Jl. Dis. Children.* 1934. Aug. Vol. 48. No. 2. pp. 359–372. With 2 figs. [32 refs.]

A general review of the subject followed by reports of six cases in California, all of whom were presumably infected with the tick-transmitted variety of relapsing fever in either Lake Tahoe region or Big Bear Valley. The symptoms in the main resemble those of the African form of the disease.

[The author's references contain numerous mistakes : Livingstone (David) is spelt without the final e ; " Sargent and Folley " presumably means Sergent and Foley ; " Hendle " is written for Hindle ; and " Manson, Philip H." instead of P. H. Manson-Bahr.] *E. H.*

Zavattari (Edoardo). Sulla asserita presenza dell' *Ornithodorus moubata* in Libia e sull' asserita conseguente presenza in Libia della febbre ricorrente da *Spirochaeta duttoni*. [**Are** *Ornithodorus moubata* **and Relapsing Fever Present in Libia?**]—*Arch. Ital. Sci. Med. Colon.* 1934. May 1. Vol. 15. No. 5. pp. 347–352. English summary (4 lines).

Gaspare (Lodato). Risposta alla nota del Prof. Zavattari " Sull' asserita presenza dell' *O. moubata* in Libia."—*Ibid.* pp. 353–359. English summary (3 lines).

Franchini (Giuseppe). A proposito di un articolo del Prof. Zavattari " Sull' asserita presenza dell' *Ornithodorus moubata* in Libia e sull' asserita conseguente presenza in Libia della febbre ricorrente da *Spirochaeta duttoni*."—*Ibid.* pp. 360–363. English summary (3 lines).

A difference of opinion as to whether *Ornithodorus moubata* occurs in Libia, Zavattari asserting that it does not and that therefore there is no danger of the spread of relapsing fever, Gaspare and Franchini maintaining the contrary. *A. G. B.*

Zimmerli (E.). **The Problem of Bronchial Spirochaetosis.**—*Tubercle.* 1934. Aug. Vol. 15. No. 11. pp. 481–497. With 2 text figs. & 10 figs. on 1 plate. [45 refs.]

A good critical review of the subject. The author's conclusions are as follows :—

" (1) *Spirochaeta bronchialis* has never been established as a species of its own, either morphologically or by experiment on the animal, or in any other way.

" (2) Spirochaetes of various shape and size may be found in any sputum, haemorrhagic or non-haemorrhagic, provided that the specimen is not washed after expectoration. In 30 out of 31 cases of pulmonary tuberculosis I have been able to demonstrate post mortem that those spirochaetes come from the mouth and are not found below the bifurcation. Therefore, the occurrence of such organisms in the sputum allows of no practical conclusions.

" (3) ' Bronchial spirochaetosis ' is not established as a disease *sui generis*, its only constant symptom according to the authors being the presence of spirochaetes in the sputum.

" (4) If a hysterical patient or a malingerer draws blood out of his gums, the product is sure to contain numerous spirochaetes : this has to be considered first in differential diagnosis to ' bronchial spirochaetosis.'

" (5) An appeal is made to our colleagues of Ceylon to state their experience in cases of chronic haemorrhagic bronchitis on that island. If such a clinical picture is common among the Cingalese, how is it characterised ? " *E. H.*

Singer (Ernst), Kotrba (Jan) & Fischl (Viktor). Die Bindung von Arzneimitteln an Spirochäten und Trypanosomen in Vitro. [**The Combination of Therapeutic Agents with Spirochaetes and Trypanosomes in Vitro.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1934. May 25. Vol. 116. No. 2. pp. 133–137. [11 refs.]

The authors have applied their micro-chemical method of analysis [*ante*, p. 510] to the examination of *Spirochaeta recurrentis* and *Trypanosoma brucei*, respectively, exposed to the action of either atoxyl or solganal *in vitro*. In addition, the effect of adding liver extract, and in the case of atoxyl other organ extracts, was tested.

The results are given in tabular form and are said to support the view that the method of action of chemotherapeutic substances is a complex phenomenon falling into three phases—(1) A physico-chemical adsorption of the substance by the cell of the pathogenic organism, (2) a change in this adsorbed substance as a result of the vital activities of the cell, resulting in the formation of an actual poison ; and (3) finally the completion of cure by the immune substances of the organism of the host.

[The figures given in the tables show such individual variations that making every allowance for difficulties of analysis, the results hardly seem sufficient to justify all the conclusions drawn from them by the authors.] *E. H.*

Fischl (Viktor) & Singer (Ernst). Gewinnung und Verhalten arzneifester Recurrensspirochäten. [**The Production and Behaviour of Drug-Fast Relapsing Fever Spirochaetes.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1934. May 25. Vol. 116. No. 2. pp. 138–145. [17 refs.]

A strain of European relapsing fever spirochaetes in mice was made resistant against solganal after only 15 passages. Mice infected with the original strain were inoculated intramuscularly with a dose of

1/10,000 gm. per 20 gm. mouse; 24 hours later the blood of the treated mouse was inoculated into normal mice and these in turn given a larger dose of the drug and the process repeated until a dose of 1/100 gm. per mouse was reached.

The properties of this solganal resistant strain were then compared with those of the original strain by exposing both to the action of various other drugs, including nine gold preparations, also neosalvarsan, and salts of rhodium and osmium. In every case the solganal-resistant strain was found to be also resistant against these other drugs, although the normal strain was susceptible. On the other hand, both strains seemed to be identical as regards their electrical properties, and also in their non-persistence in the central nervous system. Mice infected with the resistant strain were treated with two gold compounds and neosalvarsan respectively, and the plasma, blood cells, and spirochaetes subsequently analysed for the presence of gold or arsenic. In every case the spirochaetes showed no trace of these substances although one or the other was present in the plasma and red-cells. Also under such conditions normal spirochaetes have been shown to contain either gold or arsenic [*ante*, p. 510]. The results, therefore, support the view that in drug-resistant strains there is an alteration in the surface layer of the micro-organism, and the drug is no longer able to become attached to it. *E. H.*

Violle (H.). Action du ricinoléate de soude sur divers spirochètes. [**The Action of Sodium Ricinolate on Various Spirochaetes.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 25. pp. 1004–1007.

Various spirochaetes comprising *S. buccalis*, *S. ictero-haemorrhagiae*, *S. anserina*, *S. duttoni* and certain water strains were exposed to the action of solutions of sodium ricinolate. In every instance the spirochaetes were found to have been killed, as tested both by cultures and animal inoculations, even with dilutions of 1 : 1,000 in media containing proteins. *E. H.*

Brumpt (E.). Hémopéritoine chez des cobayes infectés par le spirochète de la fièvre récurrente du Turkestan. [**Haemoperitoneum in Guineapigs infected with the Spirochaete of Turkestan Relapsing Fever.**]—*Bull. Soc. Path. Exot.* 1934. June 13. Vol. 27. No. 6. pp. 510–512.

The relapsing fever of Central Asia transmitted by *Ornithodorus papillipes* [syn. *O. crossi* Brumpt], is considered to be identical with Persian relapsing fever. Consequently the name *Spirochaeta persica* Dschunkovsky 1912 has priority, and according to the author, *S. usbekistanica* Pikoul 1928, and *S. sogdianum* Nicolle and Anderson 1928, should also become synonyms. Hitherto, however, the susceptibility of guineapigs to the Persian strain has not been established. Working with a strain of relapsing fever obtained from Russian Turkestan transmitted by *O. papillipes*, and experimentally by *O. moubata*, the author has studied the pathological changes occurring in infected guineapigs, which are particularly susceptible to this strain. It was found that out of 22 infected guineapigs, 19 died, and in 10 of these the peritoneum contained a quantity of non-coagulated blood together with a large clot apparently arising from the anterior pole of the spleen. *E. H.*

SCHARRER (Berta). Ueber die Verweildauer von Hühnerspirochäten im Zentralnervensystem von Hühnern und Tauben. [**The Persistence of Fowl Spirochaetes in the Central Nervous System of Fowls and Pigeons.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1934. May 25. Vol. 116. No. 2. pp. 206–208.

The central nervous system of birds infected with fowl spirochaetosis was found to remain infected not more than 1–2 days after the blood and other organs of the body had become negative.

Of 6 fowls inoculated with a Pomeranian strain of the disease, killed respectively 1, 2, 3, 5, 9 and 31 days after the disappearance of spirochaetes from the blood, only the first fowl showed any brain infection. Similar results were obtained with four fowls infected with a Greek strain, and also with pigeons infected with these two strains and a Paris one. It would seem, therefore, that fowl spirochaetosis does not usually produce any residual infection in the central nervous system.

E. H.

SCHARRER (Berta). Ein serumfreier Nährboden für Hühnerspirochäten. [**A Serum-free Culture Medium for Fowl Spirochaetes.**]—*Zent. f. Bakt.* I. Abt. Orig. 1934. Aug. 7. Vol. 132. No. 3/4. pp. 243–244.

The author finds that the medium recommended by LI YUAN-PO for the cultivation of *S. recurrentis* [see this *Bulletin*, Vol. 30, p. 366] gives good results with the fowl spirochaete, equal to any obtained with ordinary media containing serum. Subcultures were made every 4 to 8 days and the cultures grown at 33°C. *E. H.*

MEDULLA (Candido). Nuovi casi di febbre ricorrente osservati in Cirenaica.—*Arch. Ital. Sci. Med. Colon.* 1934. May 1. Vol. 15. No. 5. pp. 335–346. With 9 figs. English summary (6 lines).

LEPTOSPIROSIS.

SCHÜFFNER (W.). **Recent Work on Leptospirosis.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1934. June 30. Vol. 28. No. 1. pp. 7–31. With 2 plates & 8 text figs. [35 refs.] (Reply to Discussion pp. 36–37. With 1 fig.)

This valuable article summarizes recent work on leptospirosis with special reference to the investigations of the author and his colleagues in Holland, and also contains the description of a divergent strain of the infective agent, *Leptospira canicola*, causing a specific canine disease.

The prevalence of Weil's disease in Holland is indicated by the fact that during the last 10 years 452 cases have been diagnosed either serologically or by isolation of leptospirae, and of these 46 died, a case fatality of 10·2 per cent. Although cases may occur in any month of the year, there is a definite accumulation of them in late summer and early autumn, the result of infection among bathers and swimmers. Details are given of the ways in which infection may be acquired and

the influence of various external factors on the leptospira. Experiments by Ruys show that these organisms can survive for at least a few hours in water of any degree of salinity, but the survival period in waters of a high degree of salinity is less than in waters with a low degree. This may explain the high incidence of the disease in Southern Holland (low salinity) compared with the much lower incidence in Northern Holland (high salinity).

All the local epidemics studied were found to be invariably accompanied by heavy murine infections (up to 56 per cent. infected) and the author considers that human infections are the result of the contamination of the water by the urine of these infected rats. It is probable, however, that infection may pass from one rat to another by way of food polluted with urine, or by sexual intercourse.

The bacteriological diagnosis of the disease is based on (*a*) detecting leptospira in freshly drawn blood by direct microscopical examination, (*b*) cultivation, (*c*) serological reaction. The first of these methods is rarely successful since even at high centrifuge speeds frequently the organisms are not precipitated. Cultivation is generally more successful when the patient's blood is collected during the first 3 or 4 days but parasites may be present as late as the 8th or 10th day. Serological tests after the disease has run its course make it possible to test the nature of suspicious infections, for the agglutinating and lytic properties of the serum persist and remain well developed for at least 8 years. Agglutination tests are best carried out with the aid of leptospira killed and preserved in 0·5 per cent. pure formalin. Suspensions of these kept for two years agglutinate in an unimpaired manner, up to the highest dilutions, and do not lyse in the same way as living organisms. Moreover, they are very convenient for the application of absorption tests.

Previously only one strain of *S. icterohaemorrhagiae* had been met with in Holland, but in November, 1931, the author cultivated a strain from the urine of a dog, which, although morphologically identical with the ordinary type, presents many points of difference. It has only a slight virulence for guineapigs; does not cause jaundice; leptospira never appear in the blood and only for a short time in the peritoneal cavity of inoculated animals; the virulence can be raised by passages through guineapigs, but even in fatal cases, produced after two or three passages, jaundice does not develop; after recovery the guineapigs almost invariably become carriers; in white mice the behaviour of the parasite is similar to that in guineapigs. Moreover, by serological tests the new strain is found to be sharply distinguished from the typical Weil strains. It has only once been identified in a human patient and hitherto has never been found in rats. In view of its divergent characters the author, in conjunction with Klarenbeek, considers it to be a distinct species, for which the name *Leptospira canicola* is proposed. *E. Hindle.*

Bagiot (Ch.) & Delbove (P.). Spirochétose ictérigène en Cochinchine. [**Spirochaetal Jaundice in Cochin China.**]—*Bull. Soc. Path. Exot.* 1934. Apr. 11. Vol. 27. No. 4. pp. 347–354. With 1 chart.

A detailed description of two cases of this disease, which in one instance gave a syndrome of " typhus hépatique " or acute infectious hepatic nephritis, with serious meningeal symptoms; whilst in the second instance there was only slight hepatitis with meningism.

During the course of the disease the serum of one of the patients agglutinated various strains of *B. proteus* X19; the agglutination, however, was only feeble and disappeared before convalescence. In both patients the blood was found to contain spirochaetes, as tested by inoculation into guineapigs, which all became infected after an incubation period of 7–10 days, and showed typical symptoms of spirochaetal jaundice.

The authors are of the opinion that this disease is probably fairly common in Cochin China, but is often overlooked owing to the difficulty of diagnosis. *E. H.*

Abe (Toshio). Ueber den Erreger des "Hasami"-Fiebers (*Spirochaeta autumnalis*). [**The Causative Agent of "Hasami" Fever** (*Spirochaeta autumnalis*).]—*Japanese Jl. Experim. Med.* 1934. June 20. Vol. 12. No. 3. pp. 255–270.

The author made a detailed study of 9 cases of this infection occurring during the autumn of 1931 in the district of Hasami, Nagasaki. The results show that the causative organism, *Spirochaeta autumnalis*, commonly referred to as "Akiyami A" strain, is serologically distinct from *S. icterohaemorrhagiae* and *S. hebdomadis* ("Akiyami B"). Guineapigs inoculated with this strain developed jaundice after an incubation period of 4 to 12 days and died one or two days later.

E. H.

Appelman (J. M.). Het isoleeren van *Leptospira icterohaemorrhagiae* uit water.—*Antonie van Leeuwenhoek Nederl. Tijdschr. v. Hyg., Microbiol. en Serol.* 1934. Vol. 1. No. 1. pp. 22–26. [*Ante*, p. 514.]

Kingo (S.). Histologische Veränderungen des Gehirns bei der Weil'schen Krankheit.—*Fukuoka Acta Med.* (*Fukuoka-Ikwadaigaku-Zasshi*). 1934. Aug. Vol. 27. No. 8. [In Japanese. German summary pp. 112–113.]

Petzetakis (M.). Ueber die Empfänglichkeit der Feldmaus für die Spirochaeta-ikterogenes-Spirochäten.—*Wien. Klin. Woch.* 1934. Aug. 24. Vol. 47. No. 34. p. 1048. [*Ante*, p. 95.]

RAT-BITE FEVER.

Levaditi (C.), Schoen (R.) & Vaisman (A.). Mode de propagation de la spirochétose provoquée par le *Spirochaeta muris*. [**The Method of Propagation of** *Spirochaeta muris* **Infection.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 25. pp. 934–937. With 2 figs.

The authors have shown previously that *Spirillum minus* in white mice can be transmitted from the mother to the offspring either *in utero* or by the ingestion of infected milk, and in the present publication extend these observations.

With regard to the prevalence of the spirillum in the mammary glands of white mice, out of 18 lactating females from Marchezais 17 contained the organism, whilst the number of uterine infections, in the

same lot, was 7 out of 15. In another series of mice from the Institut Pasteur, Paris, 3 out of 4 showed *S. minus* in the lactating mammary glands. Also a wild mouse caught in the laboratory showed a heavy infection not only in the glands, but also in the secreted milk. Young rats and mice were infected *per os* by feeding them with milk containing *S. minus*, and by killing the animals after various intervals and examining their tissues the spirilla were found to penetrate the wall of the alimentary canal within 2 to 3 hours. Infection *per os* therefore seems to be a certain method of propagating the disease among rodents. *E. Hindle.*

ARIMA (Shizuka). **Experimental Inquiry into the Transmission of Rat-Bite Fever among Rats, Part II. Successful Transmission of Rat-Bite Fever by feeding Infected Organs to White Rats.**—*Philippine Jl. Sci.* 1934. Feb. Vol. 53. No. 2. pp. 179–181.

White rats were found to be partially resistant to Manila strains of *Spirillum minus* as shown by the fact that of two rats which had been exposed to infection by ingestion, and subsequently inoculated subcutaneously with material rich in spirilla, one remained completely negative as tested both by dark ground examination of the blood and subinoculation into guineapigs. In the other rat one spirillum was found in the blood on the 42nd day and a guineapig inoculated with a suspension of its organs developed the disease.

Two rats which had been fed repeatedly on material rich in *S. minus* remained negative to microscopic examination, but their organs inoculated separately into guineapigs produced the infection. In another experiment one rat was positive and the other negative as tested in the same way. The results, therefore, suggest that rat-bite fever may be transmitted among rats by the ingestion of dead infected rats. *E. H.*

RIPLEY (Herbert S.) & VAN SANT (Helen M.). **Rat-Bite Fever acquired from a Dog.**—*Jl. Amer. Med. Assoc.* 1934. June 9. Vol. 102. No. 23. pp. 1917–1921. With 3 figs. [34 refs.]

A description of two cases of this disease apparently acquired in the laboratory from a dog.

One of these patients contracted the infection after mere contact with infected tissues, and as no skin abrasion was noted, it is suggested that the spirillum is able to penetrate the skin. Kahn tests on both patients were strongly positive and also in a dog that had been inoculated from one of them, but the Kolmer Wassermann reaction was usually negative though occasionally weakly positive. Mice, guineapigs and two dogs were infected by the inoculation of blood from the patients, and spirilla found by dark ground examination in the blood of the mice. On the other hand, the guineapigs never showed spirilla in the peripheral blood but all succumbed to the infection within two months of inoculation. A patient suffering from dementia paralytica was inoculated with infected mouse blood and developed a typical lesion and symptoms of rat-bite fever, which were terminated by treatment with arsphenamine. The author recommends a course of three to six treatments of this drug. The flagella on the spirilla were demonstrated only with Burri's india ink method. *E. H.*

Lorando (N.). Sur un cas de sodoku en Grèce. [**A Case of Sodoku in Greece.**]—*Bull. Soc. Path. Exot.* 1934. Apr. 11. Vol. 27. No. 4. pp. 304–306.

The description of a typical case of rat-bite fever following the bite of a sewer rat in Cephalonia Island. The patient recovered after a course of five injections of neosalvarsan, commencing with a dose of 0·15 gm. and gradually rising to a final dose of 0·75 gm. *E. H.*

Serefettin (O.). Un cas de sodoku à Istanbul. [**A Case of Sodoku in Istanbul.**]—*Bull. Soc. Path. Exot.* 1934. June 13. Vol. 27. No. 6. pp. 512–515.

This is the 5th case of the disease to be recorded from Istanbul, but the first in which the diagnosis was confirmed by inoculation into guinea-pigs. The clinical symptoms were somewhat atypical, consisting of 10 days fever with cutaneous eruptions and pains in various parts of the body. The patient rapidly recovered under treatment with neosalvarsan. *E. H.*

TROPICAL OPHTHALMOLOGY.

A Review of Recent Articles. XXII.*

Trachoma.—Wall[1] has drawn attention to the disease as it occurs amongst the Canadian Indians. The central and southern prairie areas and the eastern and central parts of British Columbia are the districts chiefly affected, and it is likely that the disease has been present in them for a very long time though it may have been introduced by the earlier white settlers. Changes in the habits of the people, who formerly led a healthy outdoor life but now live in badly ventilated and overcrowded huts, have increased their liability to contract the disease. Infection is mostly familial. The more elderly females wear a scarf as a head-dress and use its loose ends to wipe the eyes, nose, and cheeks of both healthy and diseased members of their family and thus freely disseminate infection. Scrofulous children are the most difficult to cure. The disease is aggravated by the dust and sandstorms met with in these dry, hot districts. Diet does not appear to influence the condition to any great extent. Education of the rising generation to co-operate in preventive hygienic measures affords the most hopeful means of eradicating the disease.

Fernando[2] has issued a timely warning against the hasty diagnosis of trachoma with respect to the population of the Philippines. He considers that true trachoma is really very rare in the islands and that the majority of the cases diagnosed as trachomatous are merely folliculosis. The inhabitants in fact appear to possess a relative immunity to the disease. Injury may result from operative interference undertaken by inexperienced medical men and nurses.

Rice, Drake & Smith[3] have given Stastnik's treatment (of copper sulphate and sodium thiosulphate, used locally and intravenously) a fair trial and failed to observe any marked beneficial effect. Large doses appeared to exercise a slight influence upon the disease, but they produced an undesirable reduction in red blood cells and haemoglobin. The authors conclude that the possible dangers of the treatment outweigh any slight benefit it may induce and it does not compare favourably with the ordinary methods. Ruata[4] advocates treatment by a vaccine called " Lacmine " prepared by him. He thinks the disease is caused by some constitutional factor acting in combination with a superimposed infection. True cicatrizing trachoma does not occur unless both factors are present in the individual. Lymphatism and acidosis together with endocrine and gastro-intestinal defects are important elements. His vaccine treatment has yielded excellent results in some of the Italian Government camps for Bedouins. The

* For the twenty-first of this series see Vol. 31, pp. 401–408.

[1] Wall (J. J.). Trachoma in the Indians of Western Canada.—*Brit. Jl. Ophthalm.* 1934. Sept. Vol. 18. No. 9. pp. 524–532.

[2] Fernando (Antonio S.). Some Remarks on Trachoma among Filipinos.—*Jl. Philippine Islands Med. Assoc.* 1934. Apr. Vol. 14. No. 4. pp. 137–141.

[3] Rice (C. E.), Drake (A. A.) & Smith (J. E.). Intravenous Use of Copper Sulphate combined with Sodium Thiosulphate in Treatment of Trachoma. —*Public Health Rep.* 1934. May 4. Vol. 49. No. 18. pp. 555–559.

[4] Ruata (V.). Two Years of Experience of Vaccine-Therapy in Trachoma and Ocular Infections.—*Jl. Egyptian Med. Assoc.* 1934. Feb. Vol. 17. No. 2. pp. 137–155.

camp inmates were particularly favourably placed with respect to their hygienic and general conditions. Lacmine is said to strengthen general resistance and not to be specific for trachoma. Acidosis is met by the hypodermic administration of a compound of sodium and potassium citrate called "Endoneutralio" WILSON[5] commenting on Ruata's paper, however, considers that the treatment is based upon false premises and states that it has been quite unsuccessful in his hands.

In the Revue Internationale du Trachome for April 1934, F. MOTAIS[6] advocates tarsal excision in the treatment of the late stages of the disease. He removes the tarsal plate through the skin surface of the lid, making an incision similar to that used in Streatfield's operation for entropion. The tarsus is shaved away until the conjunctiva is reached and care is taken not to injure that membrane. The removal of the diseased glands situated in the tarsus is wholly beneficial, and the author suggests that a study of the bacteriology and pathology of these glands might prove just as profitable as that of the trachomatous conjunctiva and follicles. ENRIQUE SELFA[7] recommends intravenous injections of citrate of copper. The injections lower the activity of heart and unstriped muscle and thus induce a fall in blood-pressure ; consequently only those patients whose circulatory and digestive systems are healthy should be subjected to the treatment. Eight centigrams of the salt are injected and the dose may be repeated after an interval of three days. MARCELL FALTA[8] contributes a useful article on the disease. He remarks how difficult is it to be sure that any two observers are discussing precisely the same disease when they refer to trachoma. He believes there is no such thing as acute trachoma ; but an acute inflammation, due to a secondary infection, may attack a trachomatous eye. He attaches much importance to ptosis as being an indication of early trachoma. The disease should be classified as mild or severe rather than early or late ; it may assume a severe form even in its earliest stage. L. POLEFF[9] discusses the preventive measures to be adopted amongst backward and mixed communities. Prophylaxis demands a government organization and must be adapted to suit the peculiar circumstances of the affected population. Special attention should be directed to the vicious circle between family and school infection.

In the issue for July 1934, MULLER[10] reviews the climatic conditions which favour the spread of the disease. Briefly these prevail in the subtropical regions where the temperature in the coldest weather does not fall below 4°C., and the humidity and rainfall are comparatively

[5] WILSON. Discussion on Dr. RUATA's Paper.—*Jl. Egyptian Med. Assoc.* 1934. Feb. Vol. 17. No. 2. pp. 156–157.

[6] MOTAIS (F.). Des effets favorables de la tarsectomie par voie cutanée pour améliorer et calmer les lésions du trachome avec ou sans entropion.—*Rev. Internat. du Trachome.* 1934. Apr. Vol. 11. No. 2. pp. 65–84. With 7 figs.

[7] SELFA (Enrique). Le citrate de cuivre dans la thérapeutique du trachome.—*Rev. Internat. du Trachome.* 1934. Apr. Vol. 11. No. 2. pp. 84–92.

[8] FALTA (Marcell). De l'unité dans quelques questions du trachome.—*Rev Internat. du Trachome.* 1934. Apr. Vol. 11. No. 2. pp. 92–99.

[9] POLEFF (L.). Quelques particularités de la lutte contre le trachome dans les pays avec une population mixte.—*Rev. Internat. du Trachome.* 1934. Apr. Vol. 11. No. 2. pp. 99–108.

[10] MULLER (A. R.). Le rôle du climat dans l'étiologie du trachome.—*Rev. Internat. du Trachome.* 1934. July. Vol. 11. No. 3. pp. 147–158. With 1 map.

low. EMANUELE GALANTE[11] describes the bio-microscopic appearance of the limbus in health and in trachoma. Normally three zones of limbic vessels are seen : (1) the palisade zone ; (1) the zone of marginal loops, and (3) the zone of end capillary loops which are empty. A primary groove separates the border of the palisade zone from the sclera and a shallow secondary groove lies between the marginal loops and the zone of end capillaries. In trachoma an excessive development of end capillary loops takes place whilst an oedema masks the grooves and causes haloes round the vessels.

Glaucoma.—The disease as it concerns the General Practitioner has been dealt with by ELLIOT[12]. His paper presents all the essential features of the disease in a manner most simple though very comprehensive. The author emphasizes the fact that eyes below the normal size are particularly liable to contract glaucoma, and states that he measures very carefully the corneal diameter of any patient who has a family history of the disease as he considers the hopefulness of the prognosis is in direct proportion to the size of the diameter.

Cataract.—KIRBY & WIENER[13] have contributed a useful review of the various factors likely to influence the occurrence of senile cataract. The authors conclude that it is difficult to limit the aetiology to any one factor or to a group of factors and are disposed to regard the disease as " a genetically induced change in the structural configuration of the lens protein molecule which makes it susceptible to the physical and chemical phenomena concerned in the production of cataract." DAY[14] reports investigations which indicate that it is possible to induce cataract formation in various animals (rats, mice, monkeys and chicks) by deprivation of vitamin G or B2. Numerous observers have previously noted in rats deprived of G the occurrence of dermatitis and loss of hair as well as ophthalmia, and their failure to notice the onset of cataract may be due to the gumming of the lids which obscures the lens changes. Cataract formation is prevented by the administration of yeast, autoclaved yeast, liver, kidney, and other foods containing vitamin G. This particular vitamin is stable to autoclaving and is soluble in water and in dilute alcohol, but is insoluble in strong alcohol. The lens change is sufficiently constant to render an ophthalmoscope an important laboratory instrument by which the earliest sign of vitamin G deprivation changes may be detected. UBALDO & AYUYAO[15] describe a technique of intracapsular extraction which they have employed on 394 eyes with very satisfactory results. They use a bridge-flap and extract the lens with a vectis introduced behind the lens from the temporal side. The authors state that posterior synechiae are present in most of their cases and they free the iris with a

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[11] GALANTE (Emanuele). Studio biomicroscopico della regione limbare dell'occhio di soggetti tracomatosi.—*Rev. Internat. du Trachome.* 1934. July. Vol. 11. No. 3. pp. 129–147. With 3 coloured figs. on 1 plate. [33 refs.] English summary.

[12] ELLIOT (R. H.). Glaucoma.—*Post Graduate Med. Jl.* 1934. Sept. Vol. 10. No. 107. pp. 323–333. With 5 figs.

[13] KIRBY (Daniel B.) & WIENER (Renée v. E-). Senile Cataract. A Study of the Biology and Chemistry of the Crystalline Lens.—*Brit. Jl. Ophthalm.* 1934. July. Vol. 18. No. 7. pp. 388–403. [25 refs.]

[14] DAY (Paul L.). Vitamin G Deficiency.—*Amer. Jl. Pub. Health.* 1934. June. Vol. 24. No. 6. Pt. 1. pp. 603–608. With 3 figs. [16 refs.]

[15] UBALDO (A. R.) & AYUYAO (C. D.). Intracapsular Extraction of Senile Cataract with Conjunctival Bridge.—*Jl. Philippine Islands Med. Assoc.* 1934. June. Vol. 14. No. 6. pp. 215–217.

spatula before introducing the loop. Vitreous prolapse occurred in only seven of their cases.

Leprosy.—Muir & Chatterji[16] strongly advocate subconjunctival injections of trypan blue in the ocular lesions of leprosy. A solution of 0·1 per cent. in normal saline is injected subconjunctivally. Repeated injections are made at suitable intervals.

Trypanosomiasis.—van den Branden & Appelmans[17] have endeavoured to determine the causes of the optic atrophy which sometimes unfortunately results from the disease or its treatment. Of 118 patients treated with tryparsamide twenty per cent. developed visual defects. Those whose cerebrospinal fluid showed pathological change were specially liable to contract ocular complications. The authors attribute the optic nerve changes to a combination of factors. These are (1) changes in the nervous system, especially the meninges, induced by the disease ; (2) the toxic action of tryparsamide, and (3) the endotoxines liberated by massive doses of the drug. Loss of vision due to atoxyl treatment is usually progressive even when treatment is stopped whereas that associated with tryparsamide may sometimes be arrested when the drug is withdrawn. Early diagnosis of the infection, so that the endotoxines liberated when treatment is instituted may be limited in amount, and a constant, careful watch for a diminution of visual acuity and visual fields are the measures most likely to prevent the occurrence of optic atrophy.

Boutonneuse fever.—Andreoli & Charlet[18] have studied the ocular complications of this disease. Naturally the eye troubles are most evident when the conjunctiva is the site of inoculation, and a condition closely resembling or identical with Parinaud's conjunctivitis then results. Very marked chemosis with non-suppurative ulceration of the conjunctiva and swelling of the pre-auricular gland occurs three days after the inoculation. Constitutional symptoms set in and the patient develops a typical boutonneuse fever. The conjunctival inflammation is limited to one eye and lasts for a variable time though it may clear in a week. The cornea remains clear throughout. A mild conjunctivitis may occur during the ordinary course of the fever. This is associated with the formation of reddish nodules on the bulbar conjunctiva and some photophobia is the chief symptom of the trouble. Iritis sometimes results from the general infection.

Syphilis.—Cuenod & Nataf[19] have reviewed very fully the influence of syphilis upon the causation of diseases of the eye in North Africa. The population is very heavily infected and the ordinary syphilitic diseases of the eye are common though the ocular complications of cerebro-spinal syphilis are remarkably rare. One of the features of ophthalmology in North Africa is that any ordinary disease of the eye such as trachoma may be greatly influenced by the co-existence of a syphilitic infection ; *e.g.*, trachoma may precipitate an attack of interstitial keratitis. It is stated that an atrophy of the optic nerve may

[16] Muir (E.) & Chatterji (S. P.). Trypan Blue in the Treatment of Leprous Eye Lesions.—*Leprosy in India.* 1934. Jan. Vol. 6. No. 1. pp. 9–11.

[17] van den Branden (F.) & Appelmans (M.). Les troubles visuels dans la trypanosomiase humaine.—*Ann. Soc. Belge de Méd. Trop.* 1934. Mar. 31. Vol. 14. No. 1. pp. 91–107. [20 refs.]

[18] Andréoli & Charlet. Fièvre boutonneuse et troubles oculaires.—*Bull. Acad. Méd.* 1934. Mar. 6. 98th Year. 3rd Ser. Vol. 111. No. 9. pp. 338–347.

[19] Cuénod & Nataf (Roger). La syphilis oculaire en Tunisie.—*Tunisie Méd.* 1934. July–Aug. Vol. 28. No. 7. pp. 415–485. [9 pages of refs.]

often be provoked by an attack of malaria or severe typhoid fever if the patient is a syphilitic. It is believed, too, that syphilis may be a cause of simple glaucoma.

Myiasis.—TRABUT[20] reports from Algeria a case of conjunctival infection by *Oestrus ovis*. The patient had been annoyed by a black fly which made persistent attempts to enter his ear. Shortly after he felt a blow on his eye and within twenty-four hours developed a catarrhal conjunctivitis with muco-purulent discharge and swollen lids. Small black specks were present in the discharge and these were identified by SERGENT as larval forms of *Oestrus ovis*.

Anaesthesia.—LYLE & FENTON[21] recommend the intravenous use of *evipan* in eye operations when a general anaesthetic is required. The solution used is 1 gram of sodium evipan in 10 cc. of distilled water, and the dose varies from 4·5 cc. in the case of old toxic patients to a full 10 cc. in strong, healthy adults. A 10 cc. syringe is used for the injection, and double the quantity of the fluid required to cause loss of consciousness (indicated by cessation of counting) is injected. A further small dose may be necessary if the above proves insufficient to produce relaxation and anaesthesia. The duration of anaesthesia is from fifteen to twenty-five minutes. Severe diseases of the liver or of the kidneys seem to be the only contra-indications. The intraocular pressure falls under evipan and this renders it especially suitable for cases of acute glaucoma. The only difficulty here is that the eyeball tends to roll up. [This may be met by the insertion of a control suture through the superior rectus.]

The Twentieth Annual Report of the Ophthalmic Section of the Egyptian Government for the year 1932[22] provides much interesting statistical information dealing as it does with nearly three-quarters of a million of new patients during the year. Thirty thousand of these suffered from gonorrhoeal ophthalmia and about seventy-five per cent. of the total from trachoma in some form. For entropion and trichiasis Snellen's operation was the one most favoured ; it accounted for 59,974 operations out of a total of 242,365. Heisrath's combined excision accounted for 330 ; senile cataract for 1,518, and trephining for 1,145. The report shows how well the Government maintains its progressive policy of medical relief. The steady fall in the number of blind persons demonstrates how successful this policy has been. *H. Kirkpatrick.*

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[20] TRABUT (G.). Un nouveau cas de myiase oculaire à *Oestrus ovis*, à Alger.—*Arch. Inst. Pasteur d'Algérie.* 1933. Dec. Vol. 11. No. 4. p. 598.

[21] LYLE (T. Keith) & FENTON (F. G.). The Advantages of Intravenous (Evipan) Anaesthesia in Ophthalmic Surgery.—*Brit. Med. Jl.* 1934. Sept. 29. pp. 589–590.

[22] EGYPT : Ministry of the Interior. Department of Public Health. Ophthalmic Section. Twentieth Annual Report of the Ophthalmic Section, for 1932.—42 pp. With 1 folding map & 1 chart. 1934. Cairo. [P.T. 10.]

LEPROSY.

Leprosy Review. 1934. July. Vol. 5. No. 3. pp. 99–148. With 6 figs. on 2 plates. Quarterly Publication of the British Empire Leprosy Relief Association, 29 Dorset Square, London, N.W.1.

This issue contains an interesting report for 1933 on the Ngomaharu Leprosy Hospital in Rhodesia by B. Moiser. After five years trial iodized and non-iodized ethyl esters of *Hydnocarpus wightiana* oil are being used exclusively in large doses up to 10 cc. intramuscularly, with or without a part intradermal. The preparation used is partly supplied by the Cape Town Government laboratory. He is clear that the results with modern treatment are now far better than he has seen in 25 years former experience. All cases should receive its benefits as some of the very worst have shown remarkable improvement, and eventually with prolonged treatment many are discharged as " apparently cured." A table of 722 treated cases shows 216, 30 per cent., thus discharged, 55 per cent. of whom were mixed or cutaneous cases. The same worker records a case of leprosy, complicated by syphilis, with great enlargement of the cervical glands from which acid-fast bacilli were discharged. Jose Rodriguez records his opinion that chaulmoogra preparations are of real value in the treatment of leprosy, but their indications and limitations require further study, and he once more points out that they do not appear to be as effective in " incipient " as in cases showing acid-fast-bacillus-containing lesions. G. R. Rao has tried ethyl esters prepared from " Pongamia Glabra," one of the oils advised in old Indian medical works together with *H. wightiana*. It had no effect on the acid-fast bacilli of the lesions, but produced intense hyperpigmentation, so may be of use in early cases of leucoderma, although it failed in advanced ones. *L. Rogers.*

Leprosy in India. 1934. July. Vol. 6. No. 3. pp. 127–171. With 4 plates & 1 fig. Issued quarterly by the Indian Council of the British Empire Leprosy Relief Association.

This number begins with an account of a promising extension of the survey-propaganda-treatment method of control by E. Muir and K. R. Chatterji. In a village union in the highly infected Bankura district a committee was formed of the local doctors and a member of each village, a leper clinic was built and treatment carried out after a survey of all the houses except those of some recalcitrant Mussulman villages. A new feature was the voluntary isolation of the highly infective cases either in huts outside the village in accordance with ancient aboriginal custom, or in the case of the well-to-do within the compound of the house, but with the door leading exteriorly only. In the survey 53 infectious and 92 non-infectious cases were found in the Hindu villages, and 16 out of the 19 highly infectious ones among poor people were thus isolated, and out of 221 known cases of leprosy 155 are attending regularly for treatment.

S. N. Chatterji reports on the decapsulation of thickened nerves, such as the ulnar and peroneal, with some improvement in sensation within about three days, but the later results are not noted. B. N. Ghosh records that an examination of 1,573 beggars in three of the divisions of Calcutta revealed 152 lepers, of whom 57 were cutaneous and 95 neural cases, doubtless mostly harmless old ones. E. Muir

reports on the relative painlessness of iodized and uniodized esters, and he concludes that the Philippine esters are rendered less painful by the addition of half per cent. iodine, but the Calcutta esters, made without distillation, are still less irritating. Reprinted reports include that of G. A. RYRIE on leprosy work in the Malay States for 1933, which records that the injection of dyes does not give lasting results, although temporary benefit follows 20 cc. doses of a 2 per cent. solution of fluorescein twice weekly in half the cases. He also notes that although universal compulsory segregation is still the legal method of dealing with leprosy, yet an out-patient clinic for ambulatory patients has become increasingly popular and the number of cases discharged after three monthly negative clinical and bacteriological examinations has increased to 151 from 91 in 1932. *L. R.*

LICHTWARDT (Hartman A.). **Leprosy in Afghanistan. A Preliminary Statement.**—*Internat. Jl. Leprosy.* Manila. 1934. Jan.–Mar. Vol. 2. No. 1. pp. 75–76.

In this brief note on thirteen years' experience at the Meshed mission hospital, Persia, it is recorded that among 200 lepers from Afghanistan all but one were in the Berberi people of Mongolian extraction, probably owing to decreased racial resistance, as the same race also suffers most in Persia. The cases are mostly advanced, but an increasing number of early cases are seen and looked after in a local voluntary leprosarium.

L. R.

RYRIE (Gordon A.). **Leprosy in Malaya.**—*Internat. Jl. Leprosy.* Manila. 1934. Jan.–Mar. Vol. 2. No. 1. pp. 77–80.

A brief account of the well-known compulsory segregation efforts to deal with leprosy in Malaya. The author thinks that under the law requiring every leper to be isolated the majority of the obvious lepers, whom he estimates at 0·1 per mille, are segregated either in the Penang Island settlement with 700 to 800 cases, or in the modern Kuala Lumpur one with about 1,000. *L. R.*

LAMPE (P. H. J.). Voordrachten over de endemiologie der lepra. [**Endemiology of Leprosy.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1934. Apr. 10 & 24. Vol. 74. Nos. 8 & 9. pp. 486–496; 522–525. With 3 graphs. English summaries.

The author emphasizes the great prevalence of leprosy in youth, with the occurrence of latent or spontaneously recovering infections and the arrest and retrogression of others. He thinks that natural resistance is not the only explanation, but that lowered resistance due to intercurrent diseases, bad conditions of life and diet deficient in vitamin A, especially in children, predispose to the disease.

In a further paper he stresses the importance of gaining the patients' confidence by avoiding all compulsion, but employing educational and treatment methods and examining all school children for leprosy.

L. R.

Soetomo & Soetopo. Bijdrage tot rationeeler bestrijding der lepra. [**Rational Control of Leprosy.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1934. May 8. Vol. 74. No. 10. pp. 606–616. [24 refs.] English summary.

These authors also lay stress on the great frequency and importance of leprosy infections during childhood and youth, often contracted by contact with adult cases, but spread among the children plays the most important part, and this fact should be the basis of control measures if they are to have the best prospects of success. *L. R.*

Rodriguez (J.) & Plantilla (F. C.). **Leprosy in Cebu. II.**—*Philippine Jl. Sci.* 1934. Jan. Vol. 53. No. 1. pp. 1–46.

The economic, hygienic and social conditions of 1,313 persons living in houses containing a leper, and of 1,817 controls have been noted in a selected poor area by house-to-house inquiries.

In the leper houses the mean ages of both sexes was higher, as were the illiteracy and the mortality with more overcrowding, less vegetation around them, dirtier habits and larger families. The diet in the leper houses included more rice and raw shellfish but less vegetables and fresh fish. Forty-seven per cent. of the bacteriologically positive lepers were under twenty years of age when the diagnosis was made, as were 57 per cent. of the " closed " or incipient cases. In 80 per cent. of the positives previous contact with a leper was noted, and 44 per cent. had lived in the same house with one; among the closed cases the corresponding figures were 68 and 3 per cent. *L. R.*

Lowe (John). **The Sex Incidence of Leprosy.**—*Internat. Jl. Leprosy.* Manila. 1934. Jan.–Mar. Vol. 2. No. 1. pp. 57–72. With 1 fig. [34 refs.]

This paper gives a good account of the literature of the subject and includes the author's experience in India. It is illustrated by a curve which brings out the very similar incidence of the disease in the two sexes during the first fifteen years of life. It shows a slightly higher rate in females at the time of puberty due to the strain of commencing sexual life, and the well-known much higher male incidence in subsequent decades after the age of twenty-five, partly due to greater promiscuity and exposure to infection. He thinks that physiological and environmental influences play their part, and he suggests that important sources of infection outside the home and family life must be operative, as in nearly all leprosy endemic areas male sufferers are about double the females, although in some countries with few cases the females are said to exceed the males. Certain resemblances between the sex incidence of tuberculosis and leprosy suggest similar predisposing factors. *L. R.*

Wade (H. W.). **Tuberculoid Changes in Leprosy. I. The Pathology of Tuberculoid Leprosy in South Africa.**—*Internat. Jl. Leprosy.* Manila. 1934. Jan.–Mar. Vol. 2. No. 1. pp. 7–38. With 52 figs. on 10 plates. [30 refs.]

This paper is a more detailed account of the naked eye and microscopical appearances of the tuberculoid cases of leprosy previously

described by the author as common in S. Africa [this *Bulletin*, Vol. 29, p. 847]. It is well illustrated by excellent photos of the skin lesions and drawings of sections; these bring out well the characteristic granular surface and linear lesions and show the giant cells, but very few lepra bacilli. The lesions often undergo complete resolution rendering this form of leprosy a mild one amenable to treatment. The paper should be read in the original by those interested. *L. R.*

CHIYUTO (Sulpicio). **Clinical Relation of the Early Macules in Children of Lepers and the Leprotic Skin Lesions in Positive Lepers.**—*Monthly Bull. Bureau of Health.* Manila. 1933. Oct. Vol. 13. No. 10. pp. 349–357.

This study deals with the same cases as were reported in a previous paper [*ante*, p. 5]. An analysis of the distribution of the early lesions shows that they were always present on the head and neck, but much less frequent on the hand than in positive lepers, but on the whole they are a replica of the skin lesions in active cases. *L. R.*

MARCHOUX (E.). Un cas d'inoculation accidentelle du bacille de Hansen en pays non lepreux. [**Case of Accidental Inoculation of Leprosy.**]—*Internat. Jl. Leprosy.* Manila. 1934. Jan.–Mar. Vol. 2. No. 1. pp. 1–6. With 2 figs. on 1 plate.

A medical assistant was pricked in a finger by a needle during the removal of a leproma and the wound was injected with a little tincture of iodine after squeezing out blood from it. Nevertheless eight years after he developed a lesion containing acid-fast bacilli at the site of the wound at a time when he was in very bad health with septicaemia, from which he died soon after; this favoured the development of the infection. *L. R.*

JORDAN (Paul). Befunde bei Lepra. I.–III.: Gleiche Hautveranderung bei Vater und Sohn. " Positive " Blutkultur. Antigen im Urin. [**Findings in Leprosy.**]—*Arch. f. Dermat. u. Syph.* 1934. July 22. Vol. 170. No. 3. pp. 365–377. With 1 fig. [35 refs.]

In the first part of this paper a case is described and illustrated showing similarly distributed depigmentation and sensibility changes in father and son. In the second part the growth of yellow colonies of acid-fast bacilli in cultures from the blood during a leprosy reaction is reported, and the third part is the record of finding a complement deviating substance in an alcoholic extract of the urine of a leper.

L. R.

MANALANG (C.). **Pathologic and Bacteriologic Survey of Lepers (Post Mortem). I.**—*Monthly Bull. Bureau of Health.* Manila. 1933. Dec. Vol. 13. No. 12. pp. 411–413.

In this short note the author records that he made microscopical examinations of the skin of the hands of 19 lepers post mortem and he succeeded in demonstrating acid-fast bacilli in 13 of them, although none had shown definite leprous lesions in this area during life. *L. R.*

Mortoatmadjo (Boentaran). De dikke-druppelmethode bij lepra-onderzoek. [**The Thick Drop Method in Leprosy.**]—*Nederl. Tijdschr. v. Geneesk.* 1934. June 23. Vol. 78. No. 25. pp. 2866–2876. English summary.

The author agrees with the conclusions of other recent workers that acid-fast bacilli found in thick drops of blood are no evidence of blood infection as they may be derived from the skin during puncture; the method generally gives negative results in nerve leprosy. *L. R.*

des Essarts (J. Quérangal) & Lefrou (G.). Contribution à l'étude histologique des lésions maculo-anesthésiques de la lèpre. [**Histology of Maculo-Anesthetic Leprosy.**]—*Bull. Soc. Path. Exot.* 1934. Apr. 11. Vol. 27. No. 4. pp. 311–318. With 2 figs.

The authors found that the microscopical examination of the skin in early maculo-anaesthetic leprosy shows characteristic changes consisting of disappearance of the papillary layer of the epidermis, which explains the loss of sensation, together with the development of clear vacuolated and vascular nodules formed of epitheloid cells with or without the presence of acid-fast bacilli; these they consider to be by themselves of diagnostic importance. *L. R.*

Natali (Claudio). Reperti istologici e patogenesi delle seguenti alterazioni nella lepra: I. Alterazioni delle ghiandole a secrezione interna. II. Ganglioneurite solare leprosa (2 casi). III. Cancro e lepra. [**Histological Changes in Leprosy.**]—*Sperimentale.* 1934. Apr.–June. Vol. 88. No. 2–3. pp. 251–324. With 21 figs. [87 refs.] English summary.

This is a lengthy and well illustrated study of the histology of two cases of nerve leprosy in which, among other things, the microscopical appearances of lesions in the thyroid, suprarenals, testicles, islands of Langerhans, and the solar plexus are described. He concludes that destructive inflammatory changes and toxo-infective action of the endocrine glands play an important part in the pathology of leprosy. *L. R.*

Lefrou (G.) & Bonnet (P.). Les modifications de l'équilibre protéique du sérum sanguin comme élément de diagnostic précoce de la lèpre. [**Changes in the Protein Equilibrium of Blood Serum and the Diagnosis of Leprosy.**]—*Bull. Soc. Path. Exot.* 1934. Apr. 11 & May 9. Vol. 27. Nos. 4 & 5. pp. 364–373; 491–500. [25 refs.]

—— & ——. Sur l'absence de relation entre la réaction de Vernes-résorcine et le déséquilibre protéique du sérum sanguin dans la lèpre.—*Ibid.* July 11. Vol. 27. No. 7. pp. 631–633.

Chorine (V.) & Prudhomme (R.). Réponse à la note de MM. Lefrou et Bonnet.—*Ibid.* pp. 633–634.

These workers in previous papers [*ante*, p. 552] found that in leprosy patients the total albumin is normal or increased, but the serum globulin is increased so that the ratio of serum albumin to serum globulin is reversed. In the first of these two papers they emphasize the diagnostic value of this blood change and consider the similar changes in other diseases possibly confused with leprosy. In febrile

states the increase in serum globulin is not usually seen, in pulmonary tuberculosis the total albumins are increased but not the globulin, in cancer they are both decreased, but in syphilis variable results are obtained. In the second paper they discuss the value of the Vernes-perethynol reaction, with the additional aid of which they hold that leprosy can be differentiated from syphilitic cases showing the same blood protein changes as leprosy, for in such cases the Vernes reaction for syphilis will be present. Illustrative cases of the value of the test are given. In the third paper Chorine and Prudhomme take exception to a statement by Lefrou and Bonnet. They point out that they have not done any work on leprosy or questioned Lefrou and Bonnet's findings. *L. R.*

Anderson (H. H.), Emerson (George) & Leake (C. D.). **A Pharmacological Evaluation of Certain Antileprotic Drugs.**—*Internat. Jl. Leprosy.* Manila. 1934. Jan.–Mar. Vol. 2. No. 1. pp. 39–55. With 3 figs. [21 refs.]

The authors hold that before any new drug is tried in the treatment of leprosy it should be tested in rat leprosy and by its action in inhibiting the growth in vitro of certain acid-fast organisms, including that of leprosy (which they believe they have cultivated) as well as by the toxicity of the preparation in animals. They record such tests in the case of the two most commonly employed chaulmoogra preparations, namely the ethyl esters and Rogers' alepol consisting mainly of sodium hydnocarpate prepared from *H. wightiana* oil, and also of three new synthetic compounds, named No. 921 (sodium chaulmoogryl p-phenetidine sulphonate), No. 923 (sodium di-hydno-chaulmoogryl p-phenetidine sulphonate, and Adams' ethyl di-N-heptyl acetate, one of the best chemically and bacteriologically of many proposed antileprotic drugs he has prepared in the course of a number of years work. Rats were used for both toxicity and therapeutic trials and *in vitro* experiments were carried out on a supposed culture of *Myco. leprae hominis* with the help of Dr. E. L. Walker and Miss M. A. Sweeney, who first showed the strong bactericidal effects of sodium hydnocarpate on acid-fast bacilli over a decade ago. The authors conclude from these important tests that the chaulmoogra ethyl esters and alepol are the most effective of the five preparations tried, and next came ethyl di-N-heptyl acetate, and last the two sulphonates. *L. R.*

Bousfield (Cyril E.). **Notes on the Treatment of Leprosy.**—*New England Jl. of Med.* 1934. May 24. Vol. 210. No. 21. pp. 1118–1120. With 2 figs.

This paper reports on the successful treatment of leprosy in a bandit-ridden part of China, where over 95 per cent. of early cases, just sufficiently advanced to allow of a certain diagnosis, are said to be curable, as are 95 per cent. moderately advanced ones recognizable at a glance and 75 per cent. of advanced cases, while the remaining incurable ones succumb to intercurrent diseases such as malaria and pneumonia. Great individual care and experience are required to enable such results to be obtained, especially in finding the optimum dose for each case. He found alepol intravenously "the least painful and generally the most effective of our whole armamentarium." It has, however, the great disadvantage of obliterating the veins in time, but

the ethyl esters intramuscularly, and especially by intradermal injection, are of great value. He also states that "the practise of the present government in China, in many places, of shooting all lepers, helps to aggravate their mental attitude." *L. R.*

Anderson (Hamilton H.), Emerson (George A.) & Fisher (B. H.). **Acute Toxicity of Trypan Blue, Gentian Violet and Brilliant Green**—*Proc. Soc. Experim. Biol. & Med.* 1934. Apr. Vol. 31. No. 7. pp. 825–828.

In view of the advocacy recently of the intravenous injection of certain aniline dyes to supplement the chaulmoogra treatment their toxicity has been tested on animals by the authors, who found that the intravenous dosage recommended by Ryrie and Ryles is within the possible lethal range as estimated by animal experiments, and they note that Ryrie admitted the occurrence of dangerous toxic effects in his patients. The intravenous toxicity of trypan blue especially varied in different animals, it depresses the central nervous system, and both brilliant green and gentian violet appear to be injurious to the liver. Moreover, in leprous rats gentian violet and brilliant green are too toxic for continued administration, but trypan blue appears to be better tolerated and to have a higher leprocidal activity. *L. R.*

Montel (M. L. R.). Un nouveau traitement de la lèpre. (Note préliminaire.) [**Treatment of Leprosy by Intravenous Methylene Blue.**]—*Bull. Soc. Méd.-Chirurg. Indochine.* 1934. Feb. Vol. 12. No. 2. pp. 147–149.

——, Le-van-Phung, Truong-van-Que & Nguyen-Ngoc-Nhuan. 2e note sur le traitement de la lèpre par les injections intraveineuses de bleu de méthylène.—*Ibid.* Mar. Vol. 12. No. 3. pp. 300–304.

——, Tran-van-Hanh, Nguyen-van-Khai, Le-van-Phung, Truong-van-Que & Nguyen-Ngoc-Nhuan. 3e note sur les injections intraveineuses de bleu de méthylène dans la lèpre : posologie, tolérance, technique.—*Ibid.* May. Vol. 12. No. 5. pp. 473–476.

These three notes deal with successive stages of a trial of intravenous injections of 1 per cent. methylene blue in continuation of a former paper [*ante*, p. 548]. In the first of the present series Montel reports that the immediate effect of the injections is better than with any other treatment, resulting in reduction in the nodules and improvement in sensation accompanied by a sense of well being. The injections were given every other day in doses gradually increased from 15 to 40 cc., and five to eleven injections per case. In the second paper it is stated that prior to the injection of the dye 4 cc. of solution containing 1 gram of urotropine was injected to favour the permeability of the methylene blue, and notes of four treated cases are given, but the dose of the dye was limited to 30 cc. to reduce the reactions that may follow it, and he advocates the use of chaulmoogra treatment in addition. In the third note he deals further with dosage and tolerance of the drug on the basis of one hundred cases and records that no reaction followed the doses in 10 per cent., in 40 per cent. fever of one or two hours duration occurred and in the remaining 50 per cent. the temperature rose to 38·5° to 39·5°C.; the higher degrees are an indication for longer intervals between the doses. He now advises commencing with a dose of 5 cc.

of the 1 per cent. solution of methylene blue and to increase by 2 to 3 cc. at a time according to tolerance, the mean full dose being 20 and 30 cc. the maximum. After 18 doses 15 to 20 days interval is allowed. The dye treatment combined with chaulmoogra preparations appears to increase the value of the latter. *L. R.*

Bouillat. L'huile de chaulmoogra, son rôle dans le traitement de la lèpre, sa fabrication à la pharmacie du gouvernement de Pondichéry. [**Chaulmoogra Oil in Leprosy : its Manufacture at Pondichery.**]—*Ann. de Méd. et de Pharm. Colon.* 1934. Jan.–Feb.–Mar. Vol. 32. No. 1. pp. 17–46. [40 refs.]

This paper first gives an historical account of the discovery of trees in various parts of the world which furnish chaulmoogra, hydnocarpus and other oils containing chaulmoogric and hydnocarpic fatty acids used in the treatment of leprosy. The method of extraction of the oil from *H. wightiana* used in the French territory of Pondichery, India, is then described as by the use of cold expression from fresh ripe seeds, which have been successfully cultivated in Pondichery as elsewhere in warm humid climates. The oil is being supplied to other French territories in Africa and elsewhere, and an estimate is made that to allow of the treatment of lepers all over the world several hundred thousand tons of the seeds may be required. Fortunately Western India alone can supply very large quantities of the oil. *L. R.*

Feng (C. T.). **The Use of Benzylephedrine as an Analgesic in Chaulmoogra Injections.**—*Chinese Med. Jl.* 1934. June. Vol. 48. No. 6. pp. 563–570. With 1 fig. [10 refs.]

After a reference to Johansen's formula of chaulmoogra oil 90 parts, olive oil 10 parts and benzocaine 3 parts to lessen the pain on intramuscular and subcutaneous injection, the author records that he prefers a mixture of chaulmoogra oil 800 cc., olive oil 200 cc. and benzylephedrine 1 gm., as the last named is readily soluble in oil and the amount required as an analgesic is only one-thirteenth of the quantity of benzocaine in the first formula. His mixture of the two oils is warmed on a water-bath to 60° to 80°C., filtered through paper while hot, one gram of pure crystals of benzylephredine is dissolved in the warm filtrate, and the whole is heated in boiling water for one hour to sterilize it before it is put up in ampoules or bottles. The melting point is about 20°C., and if warmed to body heat it will pass through a medium sized needle. The author tried it on himself up to 9 cc. doses with only very temporary pain immediately after the injection, and he states that the tolerance to it is two or three times as great as after ordinary chaulmoogra preparations. *L. R.*

Arizumi (S.). **On the Potential Transmission of *B. leprae* by Certain Insects.**—*Taiwan Igakkai Zasshi (Jl. Med. Assoc. Formosa).* 1934. Apr. Vol. 33. No. 4 (349). [In Japanese. English summary pp. 54–56.]

The author adds one more to the many investigations of the presence of acid-fast leprosy-like bacilli in insects in close contact with lepers. He found such bacilli, which he assumes to be *Myco. leprae*, in the digestive track of flies, cockroaches, and more rarely in mosquitoes, either fed on lepers or caught in wards occupied by infective cases, but the

organisms tend to die out, although some may be passed in the faeces. He thinks the cockroaches by infecting food may be the most dangerous of those investigated. *L. R.*

Lépine (P.), Markianos (J.) & Bilfinger (F.). Essais d'adaptation du bacille de Hansen à l'organisme du rat blanc. [**Attempts to adapt the Leprosy Bacillus to White Rats.**]—*Bull. Soc. Path. Exot.* 1934. Apr. 11. Vol. 27. No. 4. pp. 373–384. With 3 figs.

In these experiments leprous tissue, both from nodules and thickened macules containing acid-fast bacilli, was injected intraperitoneally with finely powdered glass to diminish the resisting powers in white mice. Extensive lesions were produced in the abdomen with spread to the lymphatic system beyond that area, and the animals became emaciated and died in about two months with numerous acid-fast bacilli in the lesions including globi, indicating their multiplication in the animals. A series of five and six passages led to a great decrease in virulence with an increasing number of bacilli losing their acid-fastness and staining blue, which the authors consider a sign of degeneration. They then lost their power of inducing pathological or lethal changes in the rats, so the object of the investigators—to cause adaptation of the human organism to rats—failed of attainment. *L. R.*

Salle (A. J.). **Acid-fast Organism from Leprous Lesions. Cultivation in Tissue Cultures and Other Mediums.**—*Jl. Infect. Dis.* 1934. May–June. Vol. 54. No. 3. pp. 347–359. With 5 figs. [25 refs.]

The author summarizes his work thus :—

" Chick tissue cultures, prepared according to the method of Carrel, were used as culture medium for the isolation of the organisms of human and rat leprosy.

" An acid-fast organism and a diphtheroid were isolated from four human nodules and one rat granuloma.

" When transfers were made to artificial culture mediums (egg, potato, agar, etc.) only the blue-staining diphtheroid multiplied.

" After the primary isolations on chick tissue cultures, minced chick embryo medium furnished an excellent substrate for the cultivation of the organisms. As with chick tissue cultures, the minced embryo medium gave rise to acid-fast and nonacid-fast organisms.

" A pure culture of the diphtheroid when inoculated into chick embryo medium gave acid-fast and nonacid-fast organisms.

" The diphtheroid and the acid-fast rods are apparently different phases of growth of the same organism.

" The organisms were acid-fast in tissues and nonacid-fast on laboratory mediums. The tinctorial characteristics varied depending on the living condition of the tissues. In vigorous actively growing tissue the organisms were strongly acid-fast. As the tissue became less vigorous, the acid-fast property was less pronounced and, finally, as the tissue died, only nonacid-fast diphtheroids were seen.

" It is believed that human and rat leprosy are caused by one and the same organism." *L. R.*

Duval (C. W.) & Holt (R. A.). **An Improved Method for in Vitro Cultivation of** *B. leprae*.—*Proc. Soc. Experim. Biol. & Med.* 1934. Apr. Vol. 31. No. 7. pp. 828–831.

The authors point out that concentration of the lepra bacillus in portions of nodules due to autolysing of the tissue cells in culture media

has been mistaken for multiplication of the organisms, which only increase as long as some tissue material remains. They therefore placed small pieces of sterile leprous tissue in 1 per cent. trypsin for 48 hours to autolyse the tissue to a butter-like consistence, and then spread it on the surface of an agar medium containing protein cleavage products, such as amino-acids, including tryptophane, cystine, tyrosine and leucine, together with sterile placenta autolysate or leucocyte extract and banana infusion with glycerine. They incubate at 37°C. in paraffin closed tubes to prevent drying, and admit fresh air every 3 to 5 days. *L. R.*

Gomes (J. M.) & Antunes (P. C. de Azevedo). **Complement Reaction in Leprosy. Activation by Potassium Iodide.**—Reprinted from *Rev. Biol. e Hig.* 1933. Dec. Vol. 4. No. 3. 3 pp. [Issued as Boletim No. 52 do Inst. de Higiene, São Paulo.]

Formerly the authors had found that with the fat free bacillus of Deycke as an antigen in lepers a negative complement fixation reaction almost always became positive after the administration of two grams of KI daily for a week. They now report some evidence to indicate that the reverse is the case with latent infections of those in contact with lepers. *L. R.*

Lowe (John). **Studies in Rat Leprosy.**—*Indian Jl. Med. Res.* 1934. July. Vol. 22. No. 1. pp. 187–198. With 8 figs. on 3 plates. [23 refs.]

The distribution and microscopical characters of the lesions of rat leprosy are described and the previous literature reviewed.

The author considers that the acid-fast bacilli of rat leprosy have not yet been successfully cultivated or generalized rat leprosy produced by their injection into those animals, and he found no evidence of the existence of a filterable virus, or of any direct action of sodium hydnocarpate on the bacillus in vitro. Rat leprosy differs from the human form in being much less limited to the skin, for it produces extensive nodular formation in the liver and spleen and much more generalized lesions of the internal organs. *L. R.*

Afanador (A.) & Bernard (P.). Evolution de la lèpre des rats après inoculations intraveineuses. [**Development of Rat Leprosy after Intravenous Inoculation.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 27. pp. 1257–1259.

——. Inoculation du bacille de Stéfansky dans les veines de rats bloqués.—*Ibid.* pp. 1260–1261.

This note deals with the lesions produced by the intravenous injection of rats with acid-fast-bacillus-containing rat leprosy material in continuation of the work already reported by Bernard [*ante*, p. 554]. The bacilli were present in the circulation at least 16 days after their injection either free or in the leucocytes of the blood. The lymphoid tissue was more involved than in natural infections, the liver showed many nodules developing in the portal spaces and containing acid-fast bacilli, and similar lesions were found in the muscle and under the endothelium of the heart, in the inter-alveolar spaces of the lung and in the thyroid and the suprarenal capsules. The skin was especially invaded and contained many bacilli. *L. R.*

Asami (Shuzo). Histopathologische Untersuchungen ueber Ratten lepra. [**Histological Investigations in Rat Leprosy.**]—*Mitt. u. Allgemeine Path. u. Path. Anat.* 1934. Vol. 8. No. 2. pp. 269–282. With 4 coloured figs. on 2 plates.

After reference to the work of Stefansky, Dean and others the author reports having found 17 rats infected with leprosy out of 2,157 examined in Japan. He describes the naked eye and microscopical changes met with in both naturally infected animals and in those inoculated with acid-fast-bacillus-containing material from other rats. These are very similar to those found by other workers, namely general infection of the glands, including the mesenteric, and nodules in the internal organs, such as the spleen, liver and lungs and in the skin. The histological changes show the reticular system to be mainly involved in the pathological process, with the formation of large cells containing numbers of the causative bacillus. *L. R.*

Landeiro (Fausto). La réaction de Rubino-Marchoux chez les lépreux. [**The Rubino-Marchoux Reaction in Lepers.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 17. pp. 174–176.

This brief note records correct results by this test in 54 of 55 leprosy cases. *L. R.*

Lefrou (G.) & Bonnet (P.). La cholestérolémie chez les lépreux de la Guadeloupe. [**Cholesteraemia in Guadeloupe Lepers.**]—*Bull. Soc. Path. Exot.* 1934. July 11. Vol. 27. No. 7. pp. 627–630.

These workers have found cholesterin estimations in lepers to give such variable results that they have given up their study as having no practical value. *L. R.*

Welch (T. B.). **Some Considerations on Diagnosis in Leprosy and on the Treatment of Lepers.**—*East African Med. Jl.* 1934. June. Vol. 11. No. 3. pp. 76–83.

This is a brief general paper on the subject and contains nothing new. The importance of diet and the treatment of complicating diseases are emphasized. *L. R.*

Montel (M. L. R.). La lèpre est-elle spontanément curable? Une observation de guérison spontanée? [**Spontaneous Case of Leprosy.**] —*Bull. Soc. Méd.-Chirurg. Indochine.* 1934. Feb. Vol. 12. No. 2. pp. 156–161.

——. Un cas de lèpre revu après un intervalle de 33 ans.—Guérison?—*Ibid.* Mar. Vol. 12. No. 3. pp. 292–297. [10 refs.]

Two cases of nerve leprosy showing the well known tendency to cessation of active symptoms and long survival with stationary nerve damage.

L.R.

Chorine (V.), Guilliny (R.) & Montestruc (E.). Inoculation de bacilles de Stéfansky dans la chambre antérieure de l'oeil du rat.—*C. R. Soc. Biol.* 1934. Vol. 116. No. 27. pp. 1261–1262.

Grillo (J.). Sobre la seroterapia antileprosa y el cultivo del bacilo de Hansen.—*Medicina Países Cálidos.* Madrid. 1934. July. Vol. 7. No. 7. pp. 305–330. [160 refs.]

Hanón (Julio L.). Anestesia segmentaria de origen leproso.—*Semana Méd.* 1934. Sept. 6. Vol. 41. No. 36 (2121). pp. 674–680. With 3 figs.

Montañes (P.). Proyecto de ficha para hacer el estudio epidemiológico establecer la profilaxis en los casos de lepra.—*Rev. San. e Hig. Publica.* 1934. Apr. Vol. 9. No. 4. pp. 320–324.

Nobl (G.). Juvenile tuberöse Lymphogranulomatose.—*Wien. Klin. Woch.* 1933. June 9. Vol. 46. No. 23. pp. 717–719. With 2 figs.

Peyri (Antonio). Epidemiología de la lepra en Cataluña.—*Rev. Méd. Barcelona.* 1934. July. Vol. 22. No. 127. pp. 8–18. With 5 figs.

Rodriguez (J.), Mabalay (E.) & Tolentino (J. C.). Gram Positive Forms of the *M. leprae* from Leprotic Lesions Bacteriologically Negative for Acid-Fast Organisms : a Preliminary Report.—*Monthly Bull. Bureau of Health.* Manila. 1933. Nov. Vol. 13. No. 11. pp. 377–389.

Santiago (Varella). A cruzada anti-anseniana no Rio Grande do Norte. (Seu inicio e actuação durante o periodo de 28 de Março de 1926 a 4 de Outubro de 1930).—*Brasil-Medico.* 1934. June 23. Vol. 48. No. 25. pp. 479–486. With 11 figs.

Vaamonde Fernandez (Joaquin). Estudio de una forma de lepra maculoan-estesica poco frecuente en Galicia.—*Medicina Países Cálidos.* Madrid. 1934. Apr. Vol. 7. No. 4. pp. 174–176. With 2 figs.

PLAGUE.

Bulletin de l'office International d'Hygiène Publique. 1934. May. Vol. 26. No. 5. pp. 830–884. With 4 maps (3 folding).—Enquête sur la peste en Afrique et sur le rôle des rongeurs sauvages et domestiques dans sa propagation.—Union de l'Afrique du Sud, Congo Belge, Afrique Occidentale Française, Colonies Britanniques et Pays de Protectorat, Egypte, Tunisie, Algérie. [**Questionary on Plague in Africa and on the Rôle of Wild and Domestic Rodents in its Propagation.**]

A questionary is a useful document from which to ascertain what are subjects worthy of investigation and those worthy of consistent record. The questionary here set out, some of the answers to which are already forthcoming, relates to plague in Africa and to the rôle of wild and domestic rodents in its propagation. It is, in the main, as follows :—

I.—*Plague among wild rodents.*—(*a*) Are there permanent signs of plague infection among wild rodents ? Does it appear to be advancing or regressing ? (*b*) Are measures for protection of towns, villages or isolated localities against infection considered possible ? Have any of these (establishment of zones free from rodents, use of hydrocyanic gas) been adopted ? What of new methods of destruction of rodents ? (*c*) Has any new knowledge been gained relating to methods used to prevent the advance of wild rodents to natural frontiers (mountains, rivers, etc.) ?

II.—*Human plague epidemic or endemic.*—Transmitted in the ordinary way (infected domestic rats, fleas, etc.). (*a*) Is it increasing or decreasing ? (*b*) Possibilities of an efficacious prophylaxis ? (*c*) Possibilities of transport of the infection by merchandise, or as a result of trade between infected zones and sea ports or neighbouring countries ? (*d*) Advantages and disadvantages of anti-plague vaccination ?

III.—*Co-operation with neighbour-territories.*—Has any opportunity occurred of special co-operation or of exchange of information with neighbouring territories in regard to any form of plague prophylaxis and, if so, what form ?

IV.—*Position of the Principal Ports in regard to Plague Infection.*—(*a*) Has the existence of rat plague been established in any port in 1932–33 and is it continuing ? This is most important for ports in countries where plague prevails in the interior.

Replies are incorporated from Union of South Africa, Belgian Congo, French East Africa, Dakar, Nigeria, Gold Coast, British Somaliland, Nyasaland, Tanganyika, Egypt, Tunis and Algeria. In many of the reports details regarding plague measures or views regarding plague transmission are given as appendices. It is surprising, however, to find that the replies to this direct and detailed questionary are mainly of the type—" The answer is in the negative." In some cases a non-committal, indirect answer is given. It is interesting to note that the only country reporting permanent signs of plague infection among wild rodents is the Union of South Africa, but that it is without sign of any important extension. This country also uses with success, and systematically, poisons and gas in the burrows of wild rodents. In Tanganyika, however, hydrocyanic gas has not been successful against gerbils.

On the whole human plague everywhere seems to be regressing, or at least to be temporarily on the decrease.

Tunis replies to the question regarding the possibilities of an efficacious prophylaxis that, " The importance of interhuman contagion has led the Public Health Service to define the routine procedure to be adopted for dealing with the propagation and dispersal of fleas. . . ." This has consisted in impregnation of the soil with a heavy gas and its treatment with tarry solutions. Temporary protection of individuals, especially of the plague personnel is obtained by the use of pyrethrine solutions. An atomizer is used for the application of the solution on approach to a case of plague.

In Tunis also transmission of plague by merchandise or by trade is not discounted, as it is by most other countries. The merchandise which is most liable to act as carrier of plague fleas is the pressed bales of rags (chiffons) which are exported.

Vaccination against plague is commended in some cases and not advocated in others. Tunis, again, has an interesting reply to this question. No antiplague vaccination has been done for two years as it is considered to be unreliable. Large scale vaccination, however, affords an opportunity of a survey and census of the population.

There is little of interest in reply to the questions on co-operation with neighbour territories or the position of the principal ports in regard to plague infection. *W. F. Harvey.*

Boyé. Au sujet du rat à mamelles multiples (*Mastomys coucha*). [**The Multimammate Rat.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. May. Vol. 26. No. 5. pp. 885–887.

One of the subjects of enquiry in the questionary of the Office International d'Hygiene Publique has reference to the rôle of certain wild rodents in the propagation of plague from one locality to another. A most interesting rodent in this connexion is what might be called the liaison rodent, half wild and half domestic, making contact at one end of its peregrinations with man and the animals around him and at the other with outlying country and truly wild animals. The multimammate rat is a type of such a rodent.

In South Africa in the towns it replaces the common mouse, lives on the remains of food and makes its nest in walls and under the ground of houses. At the same time it goes out into the fields to find other food or water. It uses the burrows deserted by gerbils for its few days absence from its village haunts and if plague gerbils have lived in these burrows the multimammate rat brings back infected fleas which it hands on to man. A communication from Senegal explains why this rodent is now called a rat instead of a mouse. It was called *Mus coucha* in 1836, then *Mus siliceus*, then *Mus microton* and recently *Mastomys coucha* by English authors. " As this animal is a rat and not a mouse it is more correct," says M. Rode, " to give it the generic name of *Epimys*, the designation for rats properly so called, since *Mus* is the designation appropriated to mice. It would become then *Epimys coucha* or white-nosed rat." Its mammae are numerous and it has at least 4 pectoral and 4 abdominal pairs ranging from the axilla to the inguinal fold. This rat is very prolific and, in Senegal, carried a minimum of 10 embryos. *W. F. H.*

Rode (P.). Contribution à l'étude des mammifères vecteurs de la peste. Détermination de quelques espèces. [**Mammalian Vectors of Plague. Determination of Some Species.**]—*Ann. de Méd. et de Pharm. Colon.* 1934. Apr.–May–June. Vol. 32. No. 2. pp. 268–272.

Body measurements, general characters and occasionally biological notes are given of the following mammalian vectors of plague :—*Xerus erythropus* (Syn. *Sciurus erythropus, Sc. albovittatus, X. leucombrinus, X. congicus*), *Arvicanthis rufinus* (Syn. *Mus rufinus*), *Epimys rattus* (*Mus rattus*) with its sub-species *Epimys rattus alexandrinus, Mastomys coucha* (Syn. *Mus coucha, M. silaceus, M. microdon*), *Mus musculus.*

The physical measurements given are :—length of head and body, length of tail, length of the posterior paw, length of the cranium, zygomatic width, post-orbital stricture and range of superior molars.

W. F. H.

Blue (Rupert). **Bubonic Plague Control in California in 1903. Origin of Ratproofing as a Control Measure.**—*California & Western Med.* 1934. May. Vol. 40. No. 5. pp. 363–365. [17 refs.]

A historical account is given in this article of what might well be called the great San Francisco Antiplague Experiment of 1903.

The view that rats and transmission of plague were closely connected had no more than made its appearance at that time and the agency of the rat flea in the dissemination of plague was not yet known. It was therefore a bold step to take for the Federal health officer in San Francisco to decide " to throw the burden upon the rat and to base the campaign upon rodent eviction and eradication. . . . Man was no longer to be held responsible for the crime." The survey made of the specially infected area showed a very insanitary condition of affairs. Whites and Chinese were living in this area in a state of overcrowding and in close association " with the Norway and other species of rodents." The rat was in possession of the field in a manner reminiscent of some Oriental cities. A series of three resolutions, which are quoted, was signed by Federal, State and City authorities and the Mercantile associations. The main tenor of these resolutions was rat destruction and a plan for rat-proofing of dwellings ; the latter is probably the first record of this as a plague measure. By the agreement drawn up the work continued to February 1905, although " no plague infection in man or rodent had been observed for a period of fourteen months." It may be taken that the measures adopted had value, for no human cases of plague were notified in San Francisco up to the summer of 1907 which was one year after the fire and earthquake. Many of the refugees had been placed in cottages with no intervening spaces between the floor and the soil. This afforded excellent harbourage for rats and both epizootic and epidemic plague made their appearance. As a counter to this the cottages were raised 18 inches from the ground and cases ceased to occur. This method has been called rat-proofing by elevation. Other methods of rat-proofing involved the use of concrete substitution for wood on ground areas, retaining walls of concrete, the sealing of hollow walls from below and the use of wire screens of proper mesh over ventilators and low windows. *W. F. H.*

KELLOGG (W. H.). **Plague.**—*California & Western Med.* 1934. Aug. Vol. 41. No. 2. pp. 103–106.

A general account of plague is given which presents all its well known features. It is in the summary that the significance of present-day plague for California is emphasized. "Whether or not the pandemic is subsiding or only following the usual history of this disease . . no one can say. The certainties we have before us are that the disease is enzootic in California and that until governmental measures of extermination of the ground squirrels are prosecuted . . . we have an ever present menace in our midst." *W. F. H.*

MEYER (K. F.). **Selvatic Plague—its Present Status in California.**—*California & Western Med.* 1934. June. Vol. 40. No. 6. pp. 407–410. With 1 fig.

Selvatic plague (JORGE. "Rongeurs et puces dans la conservation et la transmission de la peste." *Office Internat. d'Hyg. Publ.*, 1928, p. 36) is a term which is applied to that form of plague which spreads slowly from colony to colony among wild rodents, which has existed for centuries in Transbaikal and Mongolia in Asia and which, apparently, is even now establishing itself in other steppe-like or veldt-like countries. The origin of the present-day world-wide plague infection may possibly be traced to the Mongolian steppe and thence have given rise to its first real epidemic focus in Hong Kong (1894).

There seems little doubt that selvatic plague is definitely related to rat-plague and the original sequence may have been wild rodent to rat and then to man. There is evidence to-day of a reversal of the sequence and the establishment of a selvatic infection in South Africa, the Argentine and California. It is especially of the last of these foci that this article deals. There is a grave liability for this selvatic plague to "create large reservoirs of this pasteurellosis." Again a pronounced tendency of wild rodent plague is to the production of pneumonic plague in man. To this source the development of the Manchurian pneumonic epidemic of 1910 is ascribed, with its death roll of 60,000 and its 100 per cent. mortality. Some of the experimental evidence would indicate that *Past. pestis*, "isolated from wild rodents, possesses a high virulence and a definite pneumotropism."

Californian selvatic plague, which is giving much concern to the authorities, has been established through contact with the ground squirrel (*Citellus beechyi*). "Between 1903 and 1914 it has slowly but progressively invaded the coast counties south of the Sacramento River." Special investigation has been proceeding during 1933 and 1934 in the counties of California in which plague-infected squirrels have been discovered. The measures which are at present being taken to combat the prospective danger, are advice to ranchers and others of the danger attached to handling dead or sick squirrels and an active campaign—so far as funds allow—of poisoning by means of strychnine.

W. F. H.

GEORGE (P. V.) & WEBSTER (W. J.). **Plague Inquiry in the Cumbum Valley, South India.**—*Indian Jl. Med. Res.* 1934. July. Vol. 22. No. 1. pp. 77–104. With 1 map, 3 graphs & 2 plates. [19 refs.]

A restricted area, or a well defined population, may provide opportunities for the study of epidemiological theory. This is exemplified in the case of the Cumbum valley in South India, where

" climate, social conditions, rat susceptibility and specific flea population are all favourable to the spread of plague."

The observations of Reynal and Wassilieff (*ante*, p. 35) that the recognition of plague infected carcasses is most satisfactorily obtained by examination of the bone-marrow was confirmed. In the Cumbum valley there is no well marked off-season for human plague, " house rat infestation is a marked feature of life in the villages, . . . and the *cheopis* index . . . is high all the year round." There was, however, a lowering of the *cheopis* index from March to August and also a lowering of human plague incidence each year, especially in April and May. The authors consider that sufficient emphasis has not been laid upon the conditions other than a high flea index necessary for the spread of plague. " The distinction between ' infected ' and ' infective ' fleas is significant. Fewer infected fleas become infective in hot dry weather, probably meaning that fewer become ' blocked.' " Transmission of plague by fleas which have become separated, as by death, from their rat host is obviously of epidemiological importance. In this way the rat-burrow with living fleas may remain infective. Some experiments under natural conditions have demonstrated that starved infected fleas can transmit " infection after at least 29 days starvation in rat burrows." It is inferred, therefore, that gaps between epidemics may be " bridged partly by starved infected fleas." Experiments on rat destruction by means of cyanogen gas were very interesting. The preparation used was " cyanogas " and " there is strongly suggestive evidence that fumigation was of great value in the prevention of plague." That, however, this conclusion may be premature and due to a chance occurrence is admitted by the authors, who point out that " even Cumbum village practically escaped in 1928. Cyanogas was not in use then." The authors are not sanguine on the subject of the eradication of plague infection from the Cumbum valley and conclude that " anything which deprives rats of food and shelter is an antiplague measure." *W. F. H.*

i. Girard (G.) & Estrade (F.). Faits nouveaux concernant la biologie de la *X. cheopis* et son rôle dans la persistance de l'endémo-épidémie pesteuse en Emyrne. [**New Biological Facts Relating to *X. cheopis* and its Rôle in the Persistence of Endemo-epidemic Plague Conditions in Emyrna.**]—*Bull. Soc. Path. Exot.* 1934. May 9. Vol. 27. No. 5. pp. 456–458.

ii. Estrade (F.). Technique et appareil pour la capture des puces dans les poussières et débris de céréales. [**Technique and Apparatus for Capture of Fleas in the Dust of Cereals.**]—*Ibid.* pp. 458–461. With 3 figs.

i. In Antananarivo *X. cheopis* is abundant, but it is quite exceptional to meet with it inside the houses of the inhabitants. There the fleas which attack the visitor, or which can be captured, are *P. irritans* or one of the Ctenocephali. It has long been a puzzle in Madagascar what could be the mechanism of human infection. The annual recrudescence of plague was hard to explain in the absence of any apparent epizootic or an enzootic and still harder of explanation often was the connexion between murine and human plague. Recent work, however, in Ceylon and in India, confirmed in Senegal, seems to give the clue to the mystery.

The authors proceeded to investigate whether the debris of the grinding of rice might not serve as a habitat of *X. cheopis*. In this

debris these fleas *were* found. This, and the other facts collected, left not the least doubt of the interest of the observations made for the epidemiology of plague on the high plateaux of Madagascar. Round the pestle and mortar, near the storage place of the " paddy," was an abundance of bran and ground particles of rice. In this material, when taken from particular places such as obscure corners, angles of the steps of stairs, the area of the rice granary, there was no difficulty in finding *X. cheopis*. In one highly infected canton with abundance of plague cases and abandoned villages *cheopis* were collected in three villages in lots of 14, 25 and 44. Each lot when ground up and injected into a mouse and a guineapig gave rise to plague. In one of these cases only a single bloated rat had been discovered 20 days before. Paradoxical as it may seem, it had been observed previously that *X. cheopis* could never be discovered in traps even in infected houses and it had been concluded that this flea was practically non-existent outside the burrow of its normal host the rat. The real facts have now come to light.

In the discussion which followed the remarks of M. Cazanove were very pertinent. Two points struck him, the one that *cheopis* had an elective affinity for the debris and the dust of cereals, the other that infected *cheopis* can survive between annual epidemics. He thought that in this way, perhaps, certain cases of plague of irregular appearance, explainable neither by human contagion nor by rat contagion, might find their solution in the action of the infected flea alone.

ii. The apparatus which has been constructed for the capture of the elusive *X. cheopis* in rice-dust is of simple construction. It consists of a large container, a truncated cone, intended to contain water, fly-tox or alcohol. In the centre is a fixed plate with a central pin. The rest of the apparatus is built up of two parts, a funnel of hour-glass shape which fits closely over a receptacle with a 2 cm. high surrounding wall. The whole apparatus is carried to the place of investigation and the test dust is rapidly filled into the upper mouth of the funnel. The funnel with its receptacle underneath, is placed in the large container and the central pin of the fixed plate in the latter engages with the receptacle of the funnel and thus the whole is centred. Fluid is or has been placed in position in the large container ; the funnel is screwed off and removed and contact is thus established between the test dust and the fluid. As soon as this happens the dust fleas begin to jump and land in the fluid, where they are drowned and from which they can be gathered up for examination. *W. F. H.*

Jude (L. V. R.) & Lubet (J. V. H.). A propos d'une petite épidémie de peste à Beyrouth en 1932. Tentative d'une dératisation intensive. (Résultats.) [**A Small Epidemic of Plague at Beirut in 1932. Intensive Rat Campaign Results.**]—*Arch. Méd. et Pharm. Milit.* 1934. Feb. Vol. 100. No. 2. pp. 255–268.

How a small epidemic of plague at Beirut in 1932 was suppressed.

The French occupation of Syria has been followed by an enormous improvement in the sanitation of the towns of the country and of the standard of living generally. In this account of the measures adopted against plague in Beirut, a town of 200,000 inhabitants, there is a record of very determined effort. In 1920 there were 62 cases of plague and this number diminished progressively to one only in 1929. During 1930 and 1931 an increase occurred to figures of 28 and 20 respectively,

while in 1932, with a figure of 47 between April and November, it was evident that the current anti-plague methods were insufficient and that a new plan of campaign was required. The urgency of such measures was obvious if the commerce of the port and the tourist traffic were not to be affected. Quarantine regulations and sanitary measures were intensified. Vaccination was instituted on a large scale and 80,000 free inoculations were given. Something of the nature of a rat invasion was manifest at this time. One of the main anti-rat measures now begun was the use of "ratin," a Danysz virus, followed, if the total disappearance of rats did not occur in 3 weeks, by "ratinin," a Scilla poison which kills in 24 hours. The results—for it seems reasonable to call them so—of this costly activity, involving general deratization, vaccination and enforcement of sanitation was that only four human cases of plague occurred in 1933. These were sporadic and were not followed by any extension. Bacteriological examinations of captured rats now proved uniformly negative. *W. F. H.*

RAYBAUD (A.). Les rats et la peste. [**Rats and Plague.**]—*Marseilles Méd.* 1933. Dec. 25. Vol. 70. No. 36. pp. 723–732.

The experience of thirty years of plague practice in Marseilles has convinced the author that the black rat and not the grey rat is to be feared as the transmitter of the disease to man and also that deratization measures need not be as extensive as is commonly recommended.

To begin with he affirms that the formula, rat–flea–man=plague, is not to be taken as absolute dogma, for he accepts the possibility of transmission from man to man without the rat intermediary. But most of the argument is concentrated upon the danger due to the black rat. This rat came to Europe from Asia Minor in the ships of the Crusaders. It was driven out of Europe again with the advent of the grey rat. This simple fact alone accounts for the disappearance of plague from Europe and not any sensational discovery, not any improvement in the methods of general prophylaxis, not any rise in the standard of living of European peoples. A consideration of the small epidemics of plague which have occurred in Marseilles since the beginning of this century, in 1903, 1919, 1920, 1925, and 1930 form subject matter for the development of the argument on the degree of utility of deratization measures. The epizootics related to these epidemics have shown little tendency to extension, thus showing that the supposed migratory tendencies of the town rats are something of a myth. In a town like Marseilles rats live and die in the same corner of store or cellar in which they are born. How far is deratization a necessary or a reasonable measure ? asks the author. There is no question of its necessity for the rat of the ships. The ship rat is a black rat ; it is the domestic rat, the rat which lives in companionship with human beings and, perhaps most important, it can be exterminated on board ship. On the other hand extermination of the rat on shore is impossible. Its essay may be regarded as desirable by the merchant, for the rat is a thief and a spoiler of merchandise. But that is an economic, not a sanitary matter, a matter for the capitalist and not the hygienist. San Francisco tried the experiment of extermination and spent $9,000,000 before giving up this dream of perfection. It is futile to aim at perfection. A pair of rats, living on an average $2\frac{1}{2}$ years, and producing 8 young every three months would account in the end, with interbreeding, for the appearance of 2,621,440 individuals, if it were not that the forces of nature come to our assistance to restrict

this scourge, Of what avail then in restriction are the few thousands of rats killed by deratization methods ? This does not mean advocacy of abandonment, but only of the limitation of deratization methods and especially to the quarter where an epizootic focus shows itself.

Certain methods are recommended and others condemned. Rats should be captured alive with nets. Fleas leave the dead rat when cold and they are more dangerous than the living host animal. A system of careful surveillance of the rat population is recommended, with verification of the existence of infection through laboratory diagnosis. In this way a germinating focus of plague will be kept in check. Toxic baits and the use of virus are not recommended as they are apt to lead to death of the rat in its burrow. Any scheme of restricted deratization ought to involve measures to prevent the access of new rats to the places occupied by the old rats. Again insistence is made upon the fact that it is the black rat especially which, when found to be plague infected, is to be proceeded against. The black rat, dangerous as a domestic rat, is none the less a foreign rat. It may be brought to a town with merchandise and especially with merchandise that cannot be disinfested. An example of this is afforded by the trade in and entry of bananas.

In conclusion the author disclaims an attempt to negative the utility of deratization. He calls attention only to the need for discrimination in this regard, attention to the species of rat involved, the local conditions and in particular the undesirability that hygiene should confine itself to a blind acceptance of theoretical principles. *W. F. H.*

Revue de Microbiologie, d'Epidémiologie et de Parasitologie. 1934. Vol. 13. No. 1. pp. 3–88. [**Papers on Plague and Other Subjects.**] [In Russian with German summaries.]

Several articles on plague are published in this number with short summaries of their contents in German.

Dr. M. Pokrouskaja (p. 16) presents a description of what she regards as a true mutant of *Past. pestis* obtained by the action of bacteriophage on a plague culture. Its peculiarity lies in its want of virulence, for even a whole agar culture may be inoculated into the guineapig or the ziesel mouse without fatal result. Some attention is being directed at present to the use of living plague vaccine, which naturally must be non-pathogenic. This new culture should possess possibilities for such immunization and also for the production of high-value therapeutic sera.

Two articles by Chworostuchina and Grikurow respectively (pp. 34 and 49) deal with serum reactions of plague sera. In the former the use of the precipitation or flocculation reaction is advocated for titration of anti-plague sera as being more sensitive than the agglutination reaction. An antigen for the test was obtained in the filtrate from a 6-month old bouillon culture, while the testing serum was furnished by a horse immunized with living plague bacilli. In the second article a research was conducted into the agglutinating properties of the serum in ziesel mice of plague enzootic districts. It is known that the plague virus can maintain itself in the ziesel mouse during the whole of an inter-epizootic period. The author has now shown that the sera of zeisel mice obtained from different districts showed specific agglutinins in a high proportion of samples, but that these were absent in the case of young animals. The titre of the reacting sera was high

at the end of the epizootic and diminished by the beginning of the summer of the following year. It is supposed that young ziesel mice with no protective substances in the blood may form susceptible material for fresh plague outbreaks.

Two articles have reference to the viability of the plague bacillus under conditions which have a bearing on the epidemiology of the disease. E. Novikova and G. Lalazarow write on " The Preservation of Plague Virus in the Body of the Ziesel Mouse " (p. 54) and F. Semikov and I. Achourova on " The Viability of the Plague Bacillus in Grain " (p. 60). Ziesel mice were inoculated with plague and on their death they were skinned, the gastro-intestinal canal removed and the bodies left exposed to sun and air. It was found that plague bacilli could be recovered from these bodies up to 23 days. The grain experiments were intended to afford data to show when infected grain stored in elevators could be safely exported. The infection was brought about by soiling the grain with blood, faeces, urine or the orğans of plague mice. Periods of time of 1 to 8 days for one set of experiments and 12 to 27 days for another represented the viability of the plague bacillus.

Other articles deal with water bacteriology, scarlet fever toxin and anatoxin, influenza vaccination by inhalation, blood groups in the ziesel mouse, tularaemia and the helminthic fauna of the Kalmuck population. *W. F. H.*

de Figueiredo (J. M. Pacheco). Relatório dos serviços executados na peste pneumónica em Nagoá e Raçaim. [**Measures adopted against Pneumonic Plague in Nagoa and Raçaim.**]—*Arquivos da Escola Méd.-Cirurg. de Nova Goa.* Ser. B. 1933. No. 4. pp. 771–783.

An individual disembarking in Nova Goa with bubonic plague and proceeding to his own home developed secondary bronchopneumonia from which cases of primary plague pneumonia followed. An account is given of the measures of disinfection adopted, the segregation of contacts into zones according to the degree of their contact and the administration prophylactically of anti-plague serum. *W. F. H.*

Advier (M.) & Riou (M.). Bubon pesteux et lymphogranulomatose inguinale subaiguë. [**Plague Bubo and Subacute Lymphogranuloma Inguinale.**]—*Bull. Soc. Path. Exot.* 1934. Jan. 10. Vol. 27. No. 1. pp. 84–86. [Summary appears also in *Bulletin of Hygiene.*]

The authors draw attention to the possibility of certain cases of lymphogranuloma inguinale being mistaken for plague and mention eight such which they encountered in the last two outbreaks at Dakar. Conversely they saw four cases which they thought at first to be L.I. but proved to be plague. As clinical data may be inconclusive they recommend the application of all tests possible, the search for plague bacilli, the skin test of Ito-Rienstierna for chancroidal bubo and the skin test of Frei for lymphogranuloma inguinale. Naturally in case of doubt they would treat a case as one of plague pending further investigation. *L. W. Harrison.*

Estrade (M. F.). Contribution à l'étude de l'action du bactériophage dans le traitement de la peste bubonique. [**Bacteriophage in the Treatment of Bubonic Plague.**]—*Bull. Soc. Path. Exot.* 1934. June 13. Vol. 27. No. 6. pp. 609–611.

Some variation of treatment was adopted, thus giving a degree of control. The treatment ultimately preferred was to inject immediately one ampoule of bacteriophage into the bubo, follow with 40 cc. antiplague serum intravenously in the morning and another 40 cc. the same evening. This bacteriophage-serum treatment was repeated on the following day, while in severe cases as much as 120 cc. of serum was injected. A difference in results appears in the statistics, which is considered by the author to show that injection of phage into the bubo plus intravenous serum is the method of choice in bubonic plague. The figures were 5 cases of recovery out of 10 treated with serum alone intravenously, 3 out of 9 treated with phage and serum both intravenously, and 24 out of 30 treated with phage into the bubo and serum intravenously. It was found that fluid withdrawn from the bubo 24 hours after the second injection of phage was sterile. *W. F. H.*

Girard (G.). Présence d'un principe lytique très actif vis-à-vis du bacille de Yersin isolé chez des rats de Tananarive. [**A very Active Lytic Principle to the Plague Bacillus isolated from Rats of Antananarivo.**]—*Bull. Soc. Path. Exot.* 1934. May 9. Vol. 27. No. 5. p. 415. Also in *C. R. Soc. Biol.* 1934. Vol. 115. No. 11. pp. 1219–1220.

Much vain search has been made already for the presence of antiplague phage in the rats of Antananarivo. Some rats, however, were received in the laboratory with either cervical or inguinal suppurative adenitis. No plague bacilli could be found in these but in two of them a test for phage in intestinal and lymph node contents was set on foot which provided a very potent plague phage. Its activity was such that four strains of plague bacilli were lysed from the first passage and no secondary culture appeared. An idea is hazarded by the author that the presence of phage in the rats might explain the absence of plague in Antananarivo and particularly in the quarter from which the rats had come, which had been a focus for plague for ten years.

W. F. H.

Naidu (B. P. B.). **Serum Therapy of Plague.**—*Bombay Med. Jl.* 1933. Nov.–Dec. Vol. 2. Nos. 11 & 12. pp. 302–305.

A plague-serum prepared at the Haffkine Institute of Bombay was found to give more satisfactory results, when tested on animals, than the similar sera of European institutes. The present article relates to the use of this serum in two human epidemics.

In the first epidemic, which was on the wane, there were 76 cases, 43 of which were treated with sheep serum and showed a mortality of 35 per cent. while 33 were alternate-case controls with a mortality of 69·7 per cent. The " alternate cases " were, at the later stages of the epidemic, made into every third case. In the second epidemic, which was at its height, there were 179 cases treated with a buffalo serum and a mortality of 60 per cent. to compare again with alternate-case controls, numbering 167, and a mortality of 85 per cent. The sera

were injected intravenously and had no deleterious effect even in doses of 100 cc. Thus the results obtained in both epidemics are distinctly encouraging. *W. F. H.*

Bablet (J.) & Girard (G.). Lésions histologiques dans la peste pulmonaire primitive expérimentale du cobaye. [**Histological Lesions in Experimental Plague Pneumonia of the Guineapig.**]—*Ann. Inst. Pasteur.* 1934. Feb. Vol. 52. No. 2. pp. 155–165. With 4 figs.

It has been possible to produce, by the intratracheal injection of 500 million plague bacilli in 1/10 cc., a primary plague pneumonia in the guineapig.

A careful note has been made of the developing lesions. The first phase is the broncho-lymphatic, limited to the trachea, the larger bronchi and circumscribed small bronchopneumonic foci, which makes its appearance from the eighth hour after inoculation. By the commencement of the second day the second phase is entered upon and is characterized by numerous lobular foci of pneumonic reaction distributed around a central bronchiole. These foci steadily progress to suppuration, form abscesses, and are evacuated by the bronchi, but are still limited to the regional lymphatic system. A third and final phase concludes the picture. It is the short agonal stage with generalization of the lung condition and production of a bacillaemia. The abundance of plague bacilli at this stage in bronchi and trachea is exactly comparable to the condition in primary human plague pneumonia, where extreme infectivity is a feature of the disease at the approach of death. It seems justifiable for the authors to claim to have produced primary experimental plague pneumonia. They throw out a suggestion of the possibility of antiplague vaccination by intratracheal atomization of a suspension of attenuated living plague bacilli. *W. F. H.*

Girard (G.) & Robic (J.). Vaccination contre la peste au moyen d'une souche de bacilles de Yersin, vivants, de virulence atténuée. [**Vaccination against Plague with Attenuated Living Bacilli.**]—*Bull. Acad. Méd.* 1934. June 26. 98th Year. 3rd Ser. Vol. 111. No. 24. pp. 939–945.

There are many workers who maintain that vaccines if they are to be efficacious prophylactically should consist of living, although avirulent, organisms. A certain amount of hesitation in the use of such vaccines for human beings is only natural and therefore it is important that all trials of this sort should be placed on record. The authors have inoculated 15,000 persons in Madagascar with a living plague vaccine.

A large amount of animal experimentation preceded these trials, experimentation to prove the superiority of a living vaccine in immunization and the harmlessness of the procedure. Then came trials on the authors themselves, their staff and on volunteers. Finally the living vaccine from a plague strain " E.V." was used in villages where epidemic plague was prevalent. The results seem to have been notably successful for, while immunity from plague followed the use of the vaccine, the unvaccinated villages in the neighbourhood continued to be subject to the disease. No bad effects attended the inoculations either in children or in old people. The further progress of these trials will be awaited with interest. *W. F. H.*

JOUKOW-WEREJNIKOW (N.) & LIPATOVA (T.). **Immunological Studies on Plague. I. The Comparative Value of Anti-Plague Sera in the Connection with the Study of the Significance of *B. pestis* Fractions in the Pathogenicity of Plague by Means of Shwartzman's Phenomenon.**—*Rev. Microbiol., Epidémiol. et Parasit.* 1933. Vol. 12. No. 4. pp. 257–266. [15 refs.] [In Russian. English summary pp. 266–267.]

The authors have confirmed the finding that *Past. pestis* grown at 37°C., and best at pH 6·4–6·6, develops an envelope. This envelope may be isolated from the rest of the bacterial body by washing in a shaker, and demonstrated in the wash-water by a precipitin test. The antigens represented by envelope and soma respectively are different, as was shown by SCHÜTZE. Not only so but this soma is inagglutinable by the antiserum to LUSTIG's nucleoproteid, thus suggesting that the surface protein of the soma is not the same as the nucleoproteid, for the antiserum obtained by immunization with living whole bacilli agglutinates all three fractions of the bacillus.

The authors have also succeeded in obtaining Schwartzman's phenomenon (*Jl. of Exp. Med.*, 1928, Vol. 48) in rabbits " by means of the envelope culture of *B. pestis* emulsified in distilled water and lysed by subsequent freezing and thawing." The envelope substance, washed bodies of bacilli and nucleoproteid failed, when taken separately, to give the phenomenon. The bearing of these facts both upon the preparation of plague vaccines and of sera is probably well appreciated.

W. F. H.

SCHÜTZE (Harry). **The Envelope Antigen of *B. pestis* and its Antibody.**—*Brit. Jl. Experim. Path.* 1934. Aug. Vol. 15. No. 4. pp. 200–206.

The importance of envelope antigen for immunization has been demonstrated. In the case of the plague bacillus it is only fully developed with growth at 34°C. to 37°C. " It is a fragile structure, unstainable and easily dissolved . . . a large gelatinous mass . . . easily haptenized at 100°C., 15 minutes at that temperature rendering it incapable of evoking precipitins in the rabbit or of immunizing rats against a lethal dose of living plague bacilli, though it is still capable of acting unimpaired as antigen *in vitro, e.g.* with a precipitating serum." In the present study with three anti-plague therapeutic sera it has been shown that the envelope substance is chiefly contained " in the pseudo-globulin fraction of buffalo serum and in the euglobulin fraction of sheep serum." When these therapeutic sera were inoculated subcutaneously and simultaneously with a dose of living *Past. pestis* into opposite flanks of a rat, it was shown that the protection afforded was correlated with envelope content of the antigen, as recorded by the precipitin test. This envelope antigen retains little power of producing antibodies in rabbits after heating for 30 minutes at 70°C. and is completely haptenized at a temperature of 80°C. for 15 minutes. The antigen is also sensitive to alkalis and acids, being destroyed by N/8 sod. hydroxide acting for an hour at 60°C. and in 15 minutes by N/8 acetic acid at 100°C. " Plague vaccine . . . generally has a pH of over 8·0. Sterilization of the vaccine by heating to 56°C. at this reaction reduces its power of evoking precipitins in rabbits, but such

damage may be prevented by neutralizing the vaccine before sterilization. It is suggested that this sensitivity of envelope antigen to alkali may be of importance in the production of prophylactic anti-plague vaccines." *W. F. H.*

WRIGHT (Hedley D.). **The Cultivation of the Plague Bacillus.**—*Jl. Path. & Bact.* 1934. Sept. Vol. 39. No. 2. pp. 381–390. [10 refs.]

In his experiments on the conditions of optimum growth of the plague bacillus the author has found that this organism is relatively delicate, but that the fact is obscured in the ordinary methods of stock cultivation by the development of resistant forms. " The delicacy is revealed . . . by the fact that the bacillus may fail to grow from small inocula in a broth in which oxidization of the constituents has occurred on exposure to the air " and by its being readily destroyed by exposure in thin layers on an agar surface to the action of the air, particularly at 37°C." The first of these disabilities may be overcome by the addition of the requisite peptone to the nutrient medium at an early instead of a late stage in its preparation and the second if the oxygen content of the atmosphere be kept well above 1 per cent. or if serum (10 per cent.), blood (0·1 per cent.), or a reducing agent (0·05 per cent. sod. sulphite) be added to the culture medium. " Such media yield maximum growth aerobically at 37°C." *W. F. H.*

IYENGAR (M. O. T.). **Rat-Flea Survey of Peermade District, Travancore.**—*Indian Jl. Med. Res.* 1934. Apr. Vol. 21. No. 4. pp. 723–730. With 1 map.

The paper describes a flea survey made near the southern end of the Western Ghats. It is a full and careful survey on familiar lines.

The great bulk of rats trapped were *R. rattus*, on which no less than nine species of flea were found to occur. Of these, *Xenopsylla cheopis* was the commonest, but *X. astia* and several other species were numerous. *X. cheopis* was present, and its index generally above 1 and frequently above 2, in the higher parts of the district. It was completely absent from the lower parts, and this difference in altitude clearly has a considerable effect upon the other species of flea. The spot map showing the presence and absence of *cheopis* would be of greater value if the contours were marked. *P. A. Buxton.*

HART (R. W.) & PELIKAN (E. R.). **Life Span of Fleas without a Host under Normal Atmospheric Conditions occurring in Manila.**—*Public Health Rep.* 1934. June 15. Vol. 49. No. 24. pp. 699–711.

The authors wished to discover whether fleas could reach Manila alive from Calcutta or Japan, assuming that the ships were completely free of rats on which they might feed.

They took a rather limited number of fleas belonging to four different species and exposed them to the climatic conditions prevailing at the time, giving them no opportunity of feeding. They present their results *in extenso* (10 tables), but they make no distinction between the species of fleas in the tables, nor do they distinguish the sex of the fleas, which is known to have an effect on their length of life. The authors appear to have read most of what has been previously published, but

their technique is less refined than that of any of their predecessors. They conclude that, under the climatic conditions likely to prevail, the life of the flea without a host would probably not exceed 5 days, but that if the humidity were very high, it might extend to 12 days. They appear not to have considered the possibility that the fleas on a rat-free ship might feed on other animals. *P. A. Buxton.*

White (C. T.). Espèces et nombres de puces trouvées sur les rats, pris à terre, dans le port de Londres. [**Species and Number of Fleas found on Rats on Land in the Port of London.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. Feb. Vol. 26. No. 2. pp. 255–257.

Seven hundred and twenty eight rats were examined, 497 *M. rattus* and 231 *M. norvegicus*; 242 rats harboured no fleas. There were collected in all 2,246 fleas, an average of 3·08 per rat: 1,862 (83 per cent.) were *Ceratophyllus fasciatus*, 370 (16·5 per cent.) were *Leptopsylla musculi*, 9 were *Ctenophthalmus agyrtes*, and 5 *Ctenocephalus felis*. The largest number of fleas found on a rat was 61; all were *C. fasciatus*. No single *X. cheopis* or *P. irritans* was found. The curve of prevalence of fleas followed closely that of average temperature. *A. G. B.*

Antunes (Alair Accioli). A luta contra a peste no Rio de Janeiro. Serviço de desratização.—*Folha Med.* 1934. Mar. 25 & Apr. 5. Vol. 15. Nos. 9 & 10. pp. 101–104; 110–115.

Bodet (H.). Note au sujet d'un cas de mélaena chez un pesteux.—*Bull. Soc. Path. Exot.* 1934. Mar. 14. Vol. 27. No. 3. pp. 286–288.

Estrade (F.). Résultats acquis après un an de fonctionnement d'un lazaret temporaire dans la circonscription médicale de l'Emyrne.—*Bull. Soc. Path. Exot.* 1934. Mar. 14. Vol. 27. No. 3. pp. 288–289.

Estrade (F.). Conditions climatériques et peste en Emyrne.—*Bull. Soc. Path. Exot.* 1934. Apr. 11. Vol. 27. No. 4. pp. 401–403.

Hiroki (H.). Plague Epidemic at Nungan in Kirin-Province, Manchoukuo in 1933.—*Jl. Oriental Med.* 1934. Aug. Vol. 21. No. 2. pp. 20–22.

Lugowaja (L.) & Lebedewa (E.). Ueber die Kernbildungen bei *B. pestis* und *B. pseudo-tuberculosis rod. pfeifferi*.—*Giorn. di Batteriol. e Immunol.* 1934. June. Vol. 12. No. 6. pp. 1073–1081. With 36 figs. English summary (2 lines).

Jettmar (H. M.). Irrwege der Pestforschung.—*Wien. Klin. Woch.* 1934. June 1. Vol. 47. No. 22. pp. 673–675. [17 refs.]

Savino (Enrique). La campaña antipestosa del Perú y Ecuador.—*Semana Méd.* 1934. Mar. 29. Vol. 41. No. 13 (2098). pp. 992–995.

CHOLERA.

Office International d'Hygiène Publique, Paris. **Report of the Cholera Commission to the Permanent Committee of the Office International d'Hygiène Publique. Session, April-May 1934.** [M.S. copy received from the Ministry of Health, London.]

The Commission, according to this account, bases great hopes upon an enquiry to be conducted in British India under the Indian Research Fund Association with the assistance of a Cholera Consultative Committee, "the principal object of which is to discover the relation between typical cholera vibrios, their variants and the aberrant types which have been isolated from cases of cholera or pseudo-cholera and from extraneous sources. This appears to be the fundamental problem at the moment, as much from the point of view of the identification of cholera vibrios as of determining the part, if any, played by vibrios present in cholera carriers, and of the elucidation of obscure points in the epidemiology of cholera."

One of the points considered by the commission was that of the one-dose prophylactic inoculation for cholera in place of the two-dose. It is important especially where large-scale work has to be carried out among an ignorant or prejudiced population. It is always difficult to ensure the return of the individual for his second dose, but much easier to carry out a single-dose procedure. Much depends here upon the dose administered, which in various countries that had employed the single-dose ranged from 4½ to 15 thousand million vibrios: much also depends on other factors, which are still unknown or untried. We may therefore subscribe to the Commission's finding that :—"In the present state of scientific knowledge it is not possible to determine, either by experiments on laboratory animals or by the results of inoculations carried out during cholera epidemics, precisely the minimum number of vibrios necessary to ensure an adequate immunity for practical purposes; moreover, the methods of counting the number of vibrios can only give very approximate results. Furthermore, the efficacy of a vaccine depends on other factors, such as the method of preparation and the choice of strains of cholera vibrios, for which there are no definite or generally applicable rules."

The subject of the "Preparation of a Standard Agglutinating Serum for the Cholera Vibrio" is noticed below (p. 896). *W. F. Harvey.*

Morgan (M. T.). Sur la valeur de la vaccination anti-cholérique dans la pratique quarantenaire. [**Anti-Cholera Vaccination in Quarantine Practice.**]—*Bull. Office Internat. d'Hyg. Publique.* 1934. Apr. Vol. 26. No. 4. pp. 682–687. With 2 diagrams.

Data for assessment of the value of vaccination in quarantine practice are taken from the reactions to anti-cholera inoculation in producing serum antibodies.

If the presence of antibodies can be taken as a measure of immunity, it would seem clear that a real rise is not to be expected until after the fifth day from the date of inoculation, when it takes place rapidly. It is a little more rapid after a second inoculation. The duration of the immunity may be put down as not being less than one year. In quarantine practice inoculation must be considered from the point of view of the person arriving from an infected locality and not that of a person

arriving in an infected locality. As the incubation period of cholera is from 1 to 5 days, a person from an infected place may be regarded as liable to attack during the first 5 days after his departure, a risk which is much diminished if he has had inoculation more than 5 days previous to that departure. It is from the fifth day that a rise in immune bodies takes place as a result of inoculation. Such inoculated persons should be entitled to the benefit of less severe quarantine regulations than the uninoculated. And here also the difference between a person coming from an infected locality and going to an infected locality is obvious. In the latter case maximum protection is desirable and therefore two inoculations are to be preferred. In the first case, however, one inoculation only need be insisted on. *W. F. H.*

GOHAR (M. A.). **Protective Inoculation against Cholera.**—*Jl. Trop. Med. & Hyg.* 1934. Mar. 1. Vol. 37. No. 5. pp. 66–68. With 1 graph.

The results of a well organized and well controlled experiment to test the efficacy of different types of cholera vaccine are set out in this article.

Although the number of animals used in each case is rather small the survival rate is at least different for the separate vaccine series and markedly so from the control series. The types of vaccine were (1) a suspension of heat-killed and intact vibrios, (2) autolysed vibrios, and (3) a mixture of the two, each prepared from a smooth typical strain and each of the same equivalent bacterial strength. Immunization consisted in the injection subcutaneously of the dose equivalent to 500 million, 1,000 million and 5,000 million organisms at weekly intervals. Some animals succumbed to the immunization process. The testing dose was one of 2,000 million organisms given intraperitoneally and the survivals were 3 out of 11, 5 out of 13, 5 out of 12 and 0 out of 14 for the suspension of intact vibrios, the autolysed vibrios, the mixture of the two and the controls respectively. It is tentatively suggested that the mixture may produce the best result by virtue of enhancement of the action of a simple suspension from the addition of vibrio extract. *W. F. H.*

KHAN (Saranjam). **Prevention of Cholera in Rural India.**—*Indian Med. Gaz.* 1934. June. Vol. 69. No. 6. pp. 323–326. With 4 figs.

Protection of the drinking water supply from contamination is admitted to be an important anti-cholera measure and in no country more than in India. The larger towns have now municipal water supplies so that water-borne cholera is not the serious menace in them that it used to be. It is the villages of India, especially in the cholera provinces where "the chief sources of water supply are ground wells" that are now "also the chief sources of the epidemics of cholera." A device invented by the author, which is fully illustrated by drawings, is put forward as meeting village requirements. The essential requirements for any such device are :—"(*a*) It must be cheap . . . ; (*b*) it must be simple ; every village carpenter should be able to make it or repair it . . . ; and (*c*) it must be effective in protecting the well from pollution ; if bathing or washing is done near it, polluted material . . . should have no chance of getting into the well." Some further

details in the way of requirements are added, such as that :—(1) " Nobody should be able to lower his own bucket but everybody should be able to draw water when required. (2) The well must be completely covered at the top in such a way as to be easily uncovered at any time whenever it is necessary to clean the well. . . . (3) The well must be capable of being used in the ordinary way whenever so desired." [In addition to the very clear drawings one would have welcomed information on the working of an actual installation and an estimate of its actual cost.] *W. F. H.*

Morison (J.), Rice (E. Milford) & Pal Choudhury (B. K.). **Bacteriophage in the Treatment and Prevention of Cholera. A Statistical Examination.**—*Indian Jl. Med. Res.* 1934. Apr. Vol. 21. No. 4. pp. 789–907. With 9 graphs & 3 maps.

A large volume of data relating to the Assam bacteriophage trial is presented here and analysed in detail by the authors. Although it still seems impossible to obtain a presentation of a trial of any medicament on an alternate case basis with its resultant of two dissimilarly treated populations for the comparison of efficacy, there is great merit attached to this particular trial because of its large scale, the long period which is being covered, and the number of statistical experts who have scrutinized the findings claimed.

Before proceeding to give the summary of this long article, which is furnished by the authors themselves, we may pick out of the text two of their pronouncements for consideration. The test area in which bacteriophage has been applied, we may recollect, is Nowgong. " It is apparent " say the authors, " that whether we take as a control for Nowgong the distant area of Habiganj, the previous history of Nowgong itself, or the adjacent area of Darrang, the result is the same. Something has happened in Nowgong which has not happened before and has not happened in the control areas." The previous incidence of cholera in Nowgong is very strikingly compared with the incidence during the past three years of trial phage. " The death rates month by month since January 1906 have been calculated. . . . There has been no period of three consecutive years previous to 1930 in which the death rates have been comparable to those of 1930, 1931 and 1932."

Part I of this important work " deals with the prevention of cholera by the administration of cholera-dysentery bacteriophage to all cases of diarrhoea, dysentery and suspected cholera in the villages by the villagers themselves." Since 1929 epidemic cholera has been altogether absent in the experimental area, Nowgong. " Such absence of cholera during the last three and a-half years did not occur in the arbitrarily selected control area Habiganj . . . nor in the more appropriate control districts adjoining Nowgong, with which previous to 1929 there was a high correlation in the deaths from cholera." In part II outbreaks of cholera in 4 areas, each with peculiar conditions are described. Part III deals with four epidemics of cholera, in two of which bacteriophage was used while in the other two, cases " were taken to hospital and other forms of treatment were used " for comparison. Of the first two phage-epidemics it is said that " the cases treated with bacteriophage showed a mortality which, in each case, approximated a constant level. The effect of bacteriophage was as great at the beginning of the epidemic as at the end." It is interesting to learn that in one epidemic where facilities existed for the natural distribution of phage the

mortality in untreated cases showed a regular fall. In another epidemic where these facilities were non-existent the mortality remained high right into the last week of the epidemic. Part IV relates to an outbreak in which investigation was made into the subsequent cholera cases occurring in houses according to whether phage was administered to the first case or not, and demonstrated that " in groups in all other respects alike, the number of subsequent cases in houses is significantly less when the first cases in these houses received bacteriophage."

The conclusion so far arrived at by the authors is :—" The results establish a sufficient probability in favour of a significant effect of the administration of bacteriophage to form a basis of practical policy in the treatment and prevention of cholera in villages." *W. F. H.*

Aoki (K.) & Oshiro (T.). Ueber die spezifische und unspezifische Form von Choleravibrionen. [**The Specific and Non-Specific Forms of Cholera Vibrio.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1934. Sept. 18. Vol. 83. No. 3/4. pp. 291–296. [Summary appears also in *Bulletin of Hygiene.*]

Three types of cholera vibrio are recognized in Japan, the typical, the atypical and the intermediate types. No investigation appears yet to have been made into the existence of specific and non-specific antigenic components of the cholera vibrio, as in the case of other bacteria. These two components have now been obtained by setting out from one vibrio strain, preparing a serum to that strain and then testing the serum for its action upon a number of colonies of the strain. Colonies were separated which, while giving strong agglutination with the serum, showed differential characters with respect to their absorptive reactivity to the serum. These were the specific and non-specific forms, possessing specific absorptive and non-specific absorptive receptors respectively. A large number of vibrio strains were now examined for their reaction to specially prepared sera. The authors conclude from their researches that :—(1) Two serologically differentiated variation forms, specific and non-specific, are to be found among cholera vibrios and still another form in which a mixture of specific and non-specific receptors exists. (2) These 3 forms correspond with the three recognized Japanese cholera vibrio types. (3) The specific receptors are heat stable while the non-specific receptors are partly stable partly labile. (4) The three forms appear to be capable of transformation the one into the other. *W. F. H.*

Linton (Richard W.), Mitra (B. N.) & Shrivastava (D. L.). **Studies on the Antigenic Structure of** *Vibrio cholerae.* **Part V. Analyses of Vibrio Proteins : Nitrogen Distribution.**—*Indian Jl. Med. Res.* 1934. Jan. Vol. 21. No. 3. pp. 635–638.

Analysis of the protein fractions of the cholera and cholera-like vibrios was carried out by two methods, the nitrogen distribution method of van Slyke and the racemization method. The former method is the subject of communication and shows little difference between the agglutinable galactose-containing cholera vibrios and the non-agglutinable arabinose-containing water vibrios. In fact the authors' " figures do not give any indication that any marked

differences in nitrogen distribution exist among these vibrios." It is evident also " that the vibrios have a relatively simple protein structure." *W. F. H.*

Linton (Richard W.), Mitra (B. N.) & Shrivastava (D. L.). **Studies on the Antigenic Structure of *Vibrio cholerae*. Part VI. Analyses of Vibrio Proteins : Racemization.**—*Indian Jl. Med. Res.* 1934. Apr. Vol. 21. No. 4. pp. 749–760. With 2 graphs.

In this sixth part of the series of studies on the antigenic structure of the cholera vibrio it is the proteins which have been studied by the method of racemization.

The former studies of the authors have resulted in the formation of 3 groups of vibrio—Group I from clinical cholera, agglutinable and of polysaccharide I type (galactose-containing) ; Group II from clinical cholera, agglutinable and of polysaccharide II type (arabinose-containing) ; Group III isolated from water in a cholera endemic area, non-agglutinable and of polysaccharide II type (arabinose-containing). In the new studies it has been found that two proteins only were present, I and II, and that protein II shows a more rapid loss of optical activity in alkaline solutions than protein I. With the addition of this new character to the three vibrio groups mentioned, it has been found that group II has the same protein I as group I and that group III vibrios contain protein II. From the point of view of their content of polysaccharide fractions, their agglutinability and their protein content, " the vibrios of group II are truly intermediate types." The majority of vibrios from clinical cholera, however, are of group I type. A very interesting addendum states that " the fourth possible combination of the two proteins and two carbohydrates . . . has been disclosed through the analysis of an El Tor strain." The combination is, of course, polysaccharide I with protein II. *W. F. H.*

Damboviceanu (A.), Combiesco (Cornelia), Wisner (Borelly) & Soru (Eugénie). Caractérisation des vibrions cholériques par leur antigène résiduel. [**Characterization of Cholera Vibrios by their Residual Antigen.**]—*C. R. Soc. Biol.* 1934. Vol. 115. No. 9. pp. 993–995.

In this research the conclusion is reached that, " of the characters of true cholera vibrios and paracholera vibrios only the precipitating action of their residual antigens towards specific serums seems to lead to results identical with those given by specific agglutination." The extraction product obtainable from antigens in general and especially from micro-organisms, which gives no protein reaction, contains in its molecule a sugar capable of being set free by more or less prolonged hydrolysis and precipitates *in vitro* with a corresponding anti-serum, is called " residual antigen " or microbial specific polysaccharide.

Twenty-nine vibrios were investigated :—(1) Five vibrios from serious cases of cholera, agglutinating at 1/2,000, furnished residual antigens precipitating *in vitro* with anti-cholera serum. (2) Six vibrios from Iraki carriers, agglutinating at 1/2,000, did likewise. (3) Four vibrios from Basra, agglutinable at the time of isolation but becoming inagglutinable with subculture, gave residual antigens which only manifested a slight opalescence upon addition to anti-cholera serum. (4) Fourteen vibrios from El Tor, isolated upon the occurrence of a case

of cholera from pilgrims who had come from a district free of cholera for 40 years, and which were not agglutinated by anti-cholera serum, furnished residual antigens that were not precipitated by anti-cholera serum. On the strength of the results obtained the authors have come to the conclusion enunciated. *W. F. H.*

Dамвoviceanu (A.), Combiesco (Cornelia) & Soru (Eugénie). Action in vitro du bactériophage sur les propriétés du vibrion cholérique. [**Action *in Vitro* of the Bacteriophage on the Properties of the Cholera Vibrio.**]—*C. R. Soc. Biol.* 1934. Vol. 115. No. 12. pp. 1320–1322.

It was found by Doorenbos that agglutinable, lyso-sensitive, non-haemolysing vibrios could, under the action of bacteriophage *in vitro*, be transformed into non-agglutinable, lyso-resistant vibrios and that 20 per cent. became haemolytic. The experiments of the authors relate to other characters than these and have been carried out on six strains of cholera vibrio: (1) two strains, not agglutinable by trypaflavine, having a very restricted zone of agglutination by acids and a slow speed of cataphoresis, (2) two strains partially agglutinable by trypaflavine, having more extensive zones of acid agglutination and a moderate speed of cataphoresis, and (3) two strains, completely agglutinable by trypaflavine, having very extensive zones of acid agglutination and a speed of cataphoresis twice as great as the first group. Cultures of each were obtained contaminated with bacteriophage and, after the second passage, it was found that the vibrios of the first group had become strongly agglutinable by trypaflavine, their zone of acid agglutination enormously enlarged and their speed of cataphoresis twice as great. The same effects were produced on the second group but the properties of the third group underwent no change. Of the six vibrios used the two of the first group were of S type, the two of the second group appeared to be intermediate in type or mixtures, and the two of the third group were of R type. Thus the action of the bacteriophage was to transform S and intermediate types to R and to be without effect on those which were originally of R type. *W. F. H.*

Popesco-Combiesco (Cornelia) & Soru (Eugénie). Recherches sur l'agglutinabilité des vibrions cholériques et paracholériques par la trypaflavine. [**Agglutinability of Cholera and Paracholera Vibrios by Trypaflavine.**]—*C. R. Soc. Biol.* 1934. Vol. 115. No. 12. pp. 1317–1319.

An easily applied method for the separation of S and R forms of bacteria is very desirable, especially as the morphology of colonies is not sufficient for identification. It is claimed that this can be done with trypaflavine by the technique of Pampana. A 1–500 solution in 0·9 per cent. salt solution gives immediate agglutination with " rough " forms and none with pure " smooth " cultures. This method has been applied to 100 vibrios of different origin, (1) true cholera vibrios, (2) vibrios from El Tor pilgrimage of 1930, (3) vibrios from healthy carriers of the epidemic in Iraq, and (4) various paravibrios. The results obtained were (*a*) instantaneous agglutination in 26 strains, (*b*) partial agglutination in 42 strains, and (*c*) no agglutination in 31 strains. The

first group is regarded as comprising rough types, the second as a mixture and the third as a pure smooth type. These trypaflavine types agree well with those which are differentiated by means of an acid-agglutination technique. *W. F. H.*

Soru (Eugénie). Le potentiel électrique de quelques espèces de vibrions cholériques. [**Electric Potential of Some Cholera Vibrios.**] —*C. R. Soc. Biol.* 1934. Vol. 115. No. 12. pp. 1319–1320.

By these experiments the speed of cataphoresis is shown to have no relation to the classification of vibrios as true and paracholera organisms.

A close correlation is established, however, between speed of cataphoresis and the agglutination behaviour of vibrios by the trypaflavine test (see *ante*). It is explainable by the finding that organisms of R type have a higher negative electric charge than those of S type. *W. F. H.*

Yang (Y. N.) & White (P. Bruce). **Rough Variation in** *V. cholerae* **and its Relation to Resistance to Choleraphage (Type A).**—*Jl. Path. & Bact.* 1934. Mar. Vol. 38. No. 2. pp. 187–200. [10 refs.]

A special nomenclature has grown up around the subject of antigenic and phage variation which is highly technical This discussion, it is well to emphasize, " is limited to a comparison of the ultrapure vibrio and its A^r variants." An A^r variant denotes a race of vibrios resistant to A phage. A further limitation of the study has been " to heat-stable antigens of the vibrios and the corresponding antibodies." It is very interesting to learn that the authors recognize the existence of A^r races of *V. cholerae* which have not marked rough characters except their reaction with Millon's solution. In the combined study by the two authors the conclusion is reached that the process of roughening in the cholera vibrio does not differ from that studied in Salmonellas and the pneumococcus. It " involves the disappearance of a non-protein and probably carbohydrate containing substance which furnishes the characteristic O-receptor of the smooth organism." All gradations between smoothness and extreme roughness seem to exist.

Some additional experiments by one of the two authors (P. B. W.) lead to the conclusion, expressed in his own words :—" On the whole we are inclined to believe that resistance to A phage is not a modification induced by phage action but that resistant elements are present in the ultrapure culture and survive lysis." *W. F. H.*

Marras (F. M.) & Omar (Wasfy). **The Influence of Culture Media previously exposed to Ultra Violet Rays on Bacteria. Increase of Antigenic Power in** *V. cholerae* **and** *B. typhosus.*—*Jl. Egyptian Med. Assoc.* 1934. Aug. Vol. 17. No. 8. pp. 687–696.

The investigations made were into antigenic power, rapidity of growth, microscopic and cultural characters, bacterial activity and pathogenicity for three organisms, *Bact. typhosum*, *V. cholerae* and *V. El Tor* No. 67 (isolated at Tor in 1930). Antigenic power only forms the subject of this paper, measured by means of agglutination and Pfeiffer reactions and the complement fixation test. The conclusion is reached that :—" Sera obtained from rabbits inoculated with germs cultivated on agar exposed to the action of U.V. rays from a quartz

mercury vapour lamp show greater agglutinating, bacteriolytic and complement fixation powers than sera obtained by micro-organisms developed on ordinary non-irradiated agar." *W. F. H.*

Omar (Wasfy). **Cholera and Cholera-like Vibrios examined under the Filtered Ultra Violet Rays.**—*Jl. Egyptian Med. Assoc.* 1934. May. Vol. 17. No. 5. pp. 433–439.

Thick suspensions of equal opacity were made of 3 types of vibrios—(1) a non-haemolytic agglutinable cholera vibrio, (2) a haemolytic agglutinable vibrio, and (3) a haemolytic non-agglutinable vibrio. These were exposed to a pure ultra-violet beam produced by a quartz mercury vapour lamp with nickel oxide glass filter. The fluorescences given by the 3 suspensions were (1) yellowish, white and bright, (2) less bright and faint mauve, and (3) faint dirty white with a mauvish tinge. *W. F. H.*

Bulletin de l'Office International d'Hygiène Publique. 1934. July. Vol. 26. Supp. to No. 7. 86 pp. With 1 fig.—Etudes en vue de la préparation d'un sérum étalon agglutinant le vibrion cholérique. [**Studies on the Preparation of a Standard Agglutinating Cholera Serum.**]

The need for an authoritative pronouncement on serum-diagnosis of the true cholera vibrio has become urgent owing to the many epidemiological problems which are involved. It may be said, with truth, that the Mohammedan pilgrimage of the Hejaz and the quarantine camp of Tor have focussed the attention of all bacteriological laboratories, European and Oriental, upon this subject.

A beginning has been made by the Committee of the International Office of Hygiene at the instigation of the Egyptian delegate with the preparation of a standard serum. This task was entrusted to the institutes of Copenhagen and Bucharest and these " Studies " represent the results obtained by workers in various laboratories with the trial sera furnished to them. It may be well to state at the outset that this " Study . . . is far from having come to an end ; it has demonstrated some of the difficulties of the problem and raised questions which require new and delicate investigations." So much for the stage now arrived at ; its documentation is given in this supplement to the monthly Bulletin of the Office International d'Hygiène Publique. We need not enter in detail into that documentation, for it is most admirably summarized in the introduction to the monograph.

The differences in positive titre obtained to the test sera furnished to various laboratories were greater with certain strains than with others, but really qualitative divergence was exceptional. Agglutinable strains of vibrio " *appear, therefore, to belong to a fundamental type of cholera vibrio, which is the most widely distributed type and the causal agent of the majority of cases of cholera.*" A considerable number of strains, however, was recorded which were not agglutinable ; most of these strains were derived from cases of clinical cholera, but were inagglutinable by the trial sera. Several investigators referred to strains which were feebly agglutinated at the time of their isolation but which became agglutinable to titre with some months of subculture.

Other strains, again, remained permanently inagglutinable. Incidentally this term " inagglutinable " is a misnomer, for the inagglutinable vibrios are agglutinable by their own homologous sera. Former researches on this subject of inagglutinability were largely carried out before the significance of the antigenic alteration involved in the passage of an organism from the " smooth " to the " rough " (rugose) variant was appreciated. " It is possible that several of these serological individual types will ultimately prove to be only variants of the principal type," and, of course, that some of them may prove to be truly distinctive types.

One of the most important contributions to the subject of differentiation of the cholera vibrio is that of the Danish institute, which found that, next to serum reaction, the fermentation of three sugars furnished the best diagnostic method. These three sugars are saccharose, arabinose and mannose and their fermentation appears to be stable. The correlation between fermentation and agglutinability, inagglutinability and spontaneous agglutinability is close and gave, with the positive or negative fermentation result, six different vibrio types. Out of 240 " agglutinable " strains 239 gave the reaction, saccharose +, arabinose 0, mannose+ and one gave saccharose +, arabinose 0, mannose 0. At the same time 27 inagglutinable strains out of 125 and 13 spontaneously agglutinable strains out of 14 gave the same fermentation reaction as the agglutinable vibrio. The conclusion (Danish) is drawn that " the great majority of the strains examined—239—belong from the point of view of culture and serological properties to a fundamental [" principal "] type which includes all the classical strains of cholera examined." This fermentation type may then be accepted as the principal or fundamental type of the true cholera vibrio.

Other tests were investigated—the precipitation test, haemolytic power even although restricted to the blood of the goat, the cholera-red reaction and susceptibility to the currently used bacteriophage types. None of them, not even the fermentation test, " furnishes a certain diagnosis when the agglutination has not given a definite result."

To summarize :—" The greatest uncertainty continues in regard to the significance of inagglutinable vibrios isolated from clinical cases of cholera. Are they only variants of the fundamental agglutinable type . . . or are they fixed races ? Can they be grouped into serological types ? If such types exist can the disease produced by them be confused with true cholera ? What is their importance from the point of view of epidemiology ?

" In conclusion the Cholera Commission does not consider that it can at present recommend the preparation of a standard serum. . . . Research is still required and should be carried out in those countries where epidemics of cholera and cholera-like diseases occur. In those countries the points which require elucidation are :—

(*a*) What advantage is there in the use of sera prepared with antigen O [Antigen O is the antigen of the body of the vibrio, which is thermostable?]

(*b*) Are different serological types met with in true cholera and how are they to be differentiated ?

(*c*) If types can be distinguished, what is the clinical and epidemiological role to be accorded to each ? "

We may add the following excerpt from the Danish memorandum :—

" As for the El Tor strains we can assert that they do not form a homogenous biochemical and serological group. On the contrary we find in our material some El Tor strains which cannot be distinguished from classical strains of the cholera vibrio without taking into account their positive haemolytic power." *W. F. H.*

Heiberg (Borge). Des réactions de fermentation chez les vibrions. [**Fermentation Reactions of Vibrios.**]—*C. R. Soc. Biol.* 1934. Vol. 115. No. 9. pp. 984–986.

The possession of 375 different strains of vibrios has enabled the author to make out a classification of them on the basis of their fermentation reactions. He comes to the conclusion, from using 20 carbohydrates and alcohols, that three are all important, namely, the hexose mannose, the disaccharide saccharose, and the pentose l-arabinose.

After the preliminary trials on 100 strains, which determined the relative importance of the several sugars, 275 other strains were classified on the basis of fermentation of the three sugars mentioned. Group I was much the largest and was represented by vibrios fermenting saccharose and mannose in 20 hours and not fermenting arabinose. In this group there were 239 vibrios agglutinable by cholera serum, 27 non-agglutinable and 13 spontaneously agglutinable. In group II, fermenting saccharose but not fermenting either arabinose or mannose, there was only one vibrio, vibrio Dunbar, which was agglutinable, while 76 were non-agglutinable. The conclusion is drawn that, before a vibrio, especially a non-agglutinable vibrio, should be called a true cholera vibrio it must at least ferment saccharose and mannose in 20 hours and not arabinose. *W. F. H.*

Flu (P. C.). Het El-Tor vraagstuk in verband met de bacteriologische en serologische diagnostiek der choleravibrionen. [**El-Tor and Cholera Vibrios.**]—*Nederl. Tijdschr. v. Hyg., Microbiol. en Serol.* 1934. Vol. 8. No. 4. pp. 292–339. [44 refs.]

The question of what is a cholera vibrio seems to turn mainly on the point of the degree of specificity which is to be accorded to the agglutination of a test vibrio and its behaviour as regards haemolysis and haemodigestion. Flu here expresses and illustrates his well known views and draws the conclusions :—

(1) A true cholera vibrio, which was originally non-haemolytic, can acquire this character : the haemotoxin is identical with that produced by an agglutinable El-Tor vibrio.

(2) Cholera vibrios isolated from acute cases are agglutinable to or near to high-grade cholera serum titre limits, give the Pfeiffer reaction, fix complement with specific cholera serum, show haemodigestion on 10 per cent. goat blood agar and haemolyse the erythrocytes in 10 per cent. goat blood bouillon.

(3) The possibility exists that cholera vibrios which have remained a long time in the human body develop haemotoxic properties, which makes them resemble the agglutinable El-Tor vibrios. *W. F. H.*

Zimmermann (E.). Weitere Beobachtungen ueber die Hämolysine der Vibrionen. [**Further Observations on the Haemolysin of Vibrios.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1934. July 23. Vol. 82. No. 5/6. pp. 495–505.

A back and forward argumentation on the identity of the cholera and El Tor vibrios still continues. The points made by the author are that, (1) the blood agar plate is not so suitable as blood bouillon for determination of haemolysing power, and that (2) a reference to haemolysis must include the species of animal whose blood is used for test.

His culture medium for test was a blood-peptone-asparagin-ammonium lactate bouillon and the strains used were 28 cholera and 9 El-Tor, selected for special study out of a total of 112 vibrio strains. As a result of his experiments he finds that the power to haemolyse is a fixed property of vibrio strains which cannot be altered by bacteriophage action nor by passage through animals. Most of the true cholera strains can form a thermolabile human blood haemolysin. The human blood plus sheep blood haemolysing vibrios are El Tor strains and those haemolysing only human blood are cholera strains. *W. F. H.*

Shoda (T.), Koreyeda (T.) & Otomo (T.). **The Viability of Cholera Vibrios in the Human Excreta.**—*Jl. Public Health Assoc. Japan.* 1934. Apr. Vol. 10. No. 4. pp. 1–9.

The object of the experiments made by the authors was to determine the viability of cholera vibrios in human excreta under varying temperature conditions. The temperatures used were 37°C., room temperature and that of an ice-box. Under all the conditions of experimentation viability was greatest in the ice-box. Application of the results to the practice of examination for cholera carriers or cholera cases is that the material to be examined should be kept cooled.

W. F. H.

Kuroya (Masahiko) & Ono (Hirosi). **On the Types of Cholera Vibrio of the Shanghai Epidemic of 1932. (Second Report.)**—Reprinted from *Jl. Shanghai Sci. Inst.* 1933. Dec. Section 4. Vol. 1. pp. 41–68. With 1 folding plan.

As standards " the following 3 strains were used . . . Inaba (Original type), Hikojima (Middle type) and Ogawa (Variant type)." The research consisted in typing by the usual bacteriological methods and it was found that 48 strains of cholera isolated during the 1932 epidemic in Shanghai were of the " variant " type and 5 of middle type.

W. F. H.

v. Szathmáry (J.). Variabilitätsstudien an Vibrionen. [**Studies in Variability of Vibrios.**]—*Zent. f. Bakt.* I. Abt. Orig. 1934. May 7. Vol. 131. No. 5/6. pp. 366–374.

The vibrios used were water vibrios. By cultivation in the presence of rabbit erythrocytes the haemolytic power was increased, liquefaction of gelatin accelerated, coagulation of milk abolished and agglutinability increased. Passage by the human intestine, effected by injection into the mucosa of portions obtained from the pathologist, produced certain changes. These were :—abolition of the power to coagulate milk, delay in liquefaction of gelatin, first delay and then abolition of the liquefaction of coagulated blood serum. Precipitability of metallic salts was increased and power of capillary rise was lowered. No adaptation to the human intestine could be demonstrated. *W. F. H.*

PELLAGRA.

Sinaï (I.). Contributo all'epidemiologia della pellagra in Costantinopoli. [**Epidemiology of Pellagra in Constantinople.**]—*Ann. d'Igiene.* 1934. Mar. Vol. 44. No. 3. pp. 245–247.

An account is given of pellagra in Constantinople.

During 17 years of practice in Constantinople the author has seen many cases of pellagra. In 1931 the number of cases increased, and the disease was of greater severity, with frequently a fatal termination. Nearly all the patients were Jews, and the disease seemed to pick out members of this race even when they lived in scattered groups among the Mahommedans, who themselves escaped. Though most of the cases were poor, there seemed to be no definite quantitative food deficiency. The consumption of unleavened bread was regarded as the only probable causative factor. Unleavened bread is eaten by the Jewish community during the eight days of Passover, and during the following month (*i.e.*, about Easter). Formerly the sale of this bread was under religious control, and only the best grain was used. Now, however, there are no restrictions, and it happens that unsold stocks of the previous year are sold as fresh in the following spring. The author suggests that possibly a germ develops in the stored flour and thus gives rise to pellagra.

[Though an organismal factor is suggested by the author, it is possible that the absence of yeast in the bread may be of importance. I am informed that in strict Jewish families no leaven, *i.e.*, no yeast, is allowed in the house during the eight days of Passover. Afterwards many Jews continue to eat unleavened bread as a delicacy, but not to the exclusion of leavened bread.] *A. D. Bigland.*

Bredmose (Georg V.). Undersøgelser over pellagras forekomst paa Viborg sindssygehospital i aarene 1924–33. [**A Study of the Incidence of Pellagra in the Viborg Asylum in the Years 1924–33.**] —*Hospitalstidende.* 1934. June 12. Vol. 77. No. 24. pp. 694–702. [14 refs.]

The recent recognition of pellagra as an asylum disease led late in 1933 to a search for cases of this disease in the Danish Government asylums, several of which have yielded a modest crop of such cases. Bredmose's investigations cover the 10-year period 1924–33. A scrutiny of the records revealed 7 cases, the earliest being observed in 1927.

There was no chronological uniformity in the appearance of the various symptoms, with this exception that the changes in the skin invariably occurred after the onset of the diarrhoea. The last 2 cases were so recent that the correct diagnosis led to the institution of specific treatment with vitamin B_2 foods and preparations. In both these cases the pigmentation and hyperkeratosis reacted satisfactorily, disappearing altogether in one case. Various other manifestations of pellagra diminished under this treatment, but there was no improvement in the mental condition. A relative lymphocytosis was found in 3 cases and spinal symptoms in 5. It would seem that secondary pellagra may occur in any form of mental disease, and that a special pellagra psychosis is not demonstrable. The patient who suffers from protracted intestinal disease or who refuses to eat is liable to contract

pellagra, the incidence of which in the Danish asylums is somewhere between 3 and 10 per thousand. With timely and appropriate treatment, the prognosis ought not to be bad. *C. Lillingston.*

Flinker (Robert). Die Lokalisation der pellagrösen Hautveränderungen. [**Localization of the Skin Changes in Pellagra.**]—*Schweiz. Med. Woch.* 1934. Feb. 17. No. 7. pp. 150–153. With 1 fig. [21 refs.]

A description of the usual sites of dermatitis in pellagra is given. It is pointed out that not only light or pressure, but any irritant applied to the skin during the eruptive stage of the disease will cause dermatitis.

It used to be thought, and still is by holders of the photo-dynamic theory, that the skin eruption in pellagra occurs under the influence of light. The author has kept under observation some 60 pellagrins for the purpose of studying the skin localization of the disease. It was found that in addition to the typically situated rash, there were two cases of generalized branny desquamation with pigmentation, and in four cases the same appearances were localized to the abdomen and loins. Stress is laid upon the importance of the rash occurring over pressure areas. In addition to such factors as light and pressure, at a certain stage of the disease any source of irritation wherever applied produces a typical pellagrous eruption. Thus, in two cases during the early part of the natural eruption stage, the application of a mustard leaf for one hour a day (in one instance on the chest, and in another on the loin) caused a typical dermatitis in a few days. In a third case, showing more chronic skin changes, the mustard leaf caused a reaction if anything less marked than that produced in a normal control. Thus it will be seen that a chemical irritant will produce typical dermatitis when applied to the skin of pellagrins during the ingravescent stage of the natural eruption. Any area of the skin surface can be so affected.

A. D. B.

Sabry (Ibrahim). **On the Intimate Relationship between Pellagra and Pemphigus.**—*Jl. Trop. Med. & Hyg.* 1934. Aug. 1. Vol. 37. No. 15. pp. 225–231. Also in *Jl. Egyptian Med. Assoc.* 1934. Sept. Vol. 17. No. 9. pp. 755–772.

The similarity between the different types of pellagra and pemphigus is pointed out, and the possibility of both diseases being due to the same toxin, dopa, is suggested.

The author has already put forward his view that pellagra is caused by the toxin, " dioxyphenylalanine," or dopa [see this *Bulletin*, Vol. 30, p. 149]. He has also suggested that this toxic dopa is oxidized by the basal cells of the skin to non-toxic melanin, and that the hyperpigmentation of pellagra is Nature's way of neutralizing the poison and overcoming the disease.

Pellagra and pemphigus have always been regarded as two distinct diseases on account of the differences in skin changes and geographical distribution. Nevertheless the author was struck by the similarity between some cases of pellagra and pemphigus. He points out that the excessive melanin formation about the skin lesions seen in pemphigus vegetans is identical with that found in pellagra, and he suggests

that both diseases are associated with the presence of the same toxin, dopa. In support of this view the following points are stressed :—

(1) Both diseases present different types and varying degrees of malignancy. " The wide variations observed in the severity and degree of malignancy in one and the same disease are variations much wider than those existing between certain types of pellagra on the one hand, and certain types of pemphigus on the other hand."

The differences between the two diseases may be due either to slight alterations in the causal toxin or to individual or racial peculiarities.

(2) Both diseases tend to affect the skin, the gastro-intestinal system and the nervous system. The changes in the last two systems, it is claimed, are almost identical in corresponding types of both diseases.

(3) There is a close correspondence between the clinical types of both conditions, especially in the acute malignant forms.

The author is of the opinion, after lengthy discussion and argument, that the skin lesions of pellagra are not so very different from those met with in pemphigus. He has been struck by the fact that most of the pemphigus cases in temperate zones occur among the Jews or " persons of otherwise oriental (*i.e.*, tropical) ancestry," and he comes to the conclusion that " pemphigus is pellagra in disguise ; pellagra moulded by northern environments." When the skin is naturally resistant, as it is in tropical races, the response to dopa is the hyperpigmentation of pellagra and, conversely, the response in less resistant skins is of the nature of pemphigus. The various ways in which a toxin such as arsenic affects the skin (pigmentation, bullae formation, etc.) is a very interesting point in this respect and the possibility of its action upon the adrenal glands is suggested.

Four cases of pemphigus and one case of " malignant pemphigus-pellagra " were treated by the author's sodium thiosulphate method (daily intravenous injections of 10 cc. of a 10 per cent. solution). Two cases made a good recovery and three died. *A. D. B.*

Wheeler (G. A.) & Hunt (D. J.). **The Pellagra-preventive Value of Green Onions, Lettuce Leaves, Pork Shoulder, and Peanut Meal.**—*Public Health Rep.* 1934. June 22. Vol. 49. No. 25. pp. 732–736.

The authors continue their studies dealing with the pellagra-preventive value of various foodstuffs.

Canned green onions and canned lettuce leaves are found to be poor in P.P. factor, while lean pork shoulder and peanut meal are good sources of this accessory. *A. D. B.*

Sebrell (W. H.). **Table showing the Pellagra-preventive Value of Various Foods.**—*Public Health Rep.* 1934. June 29. Vol. 49. No. 26. pp. 754–756. [18 refs.]

A full table of various foodstuffs, showing the pellagra-preventive value of each, is given. This table is intended to be used in the treatment and prevention of pellagra, and only those foods are included which have been tested in both human beings and dogs. The foods are graded for this purpose into four groups : " Good," " Fair," " Slight," and " None." It is important to note that these gradings refer only to the actual daily amount of the food given in the table.

The list will be found to be most useful by those called upon to deal with the treatment and prevention of pellagra, but it is unfortunately impossible to include the actual table in a short abstract. *A. D. B.*

GUILLAIN (G.), BERTRAND (I.), MOLLARET (P.) & LEREBOULLET (J.). Étude anatomique d'un cas français de pellagre avec paraplégie. [**Anatomical Study of a Case of Pellagra with Paraplegia.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1934. May 21. 50th Year. 3rd Ser. No. 16. pp. 650–657. With 7 figs.

This contains a detailed account of the neuro-histological findings in a case of pellagra occurring in France.

At a meeting of the Société Médicale des Hôpitaux de Paris on March 27th 1931, a case of pellagra in a Frenchman aged 56 was shown. In addition to skin manifestations a spastic paraplegia had been present for six months. Under treatment the paraplegia improved for a time, but later it increased again and soon became complete. Oedema of the legs and perforating ulcers of the feet were noted. The trunk, arms, cranial nerves and mentality remained normal throughout. Phthisis supervened and the patient died on November 30th, 1932.

A detailed post-mortem examination of the nervous system was made. The following are among the important findings :—The brain as a whole appeared normal, but the spinal cord showed extremely widespread lesions, involving all the segments and attacking the posterior and antero-lateral columns to a variable degree. A full account is given of the appearance of the various tracts at different spinal levels and those particularly interested in the subject should consult the original paper. Some of the anterior horn cells in the cervical region showed atrophic changes with alterations in pigment distribution and nuclear position. All the ganglion cells were fully charged with lipo-pigment. Slight secondary changes in the neuroglia were present and the vessels showed no endarteritis, though in a number of capillaries hyaline thickening of the wall external to the endothelium was observed. Perivascular infiltration was absent. The Betz cells in the cerebrum, according to some workers affected in pellagra psychoses, were normal in this case and in the peripheral nerves no myelin changes could be demonstrated. The products of disintegration in the spinal cord were found to be essentially of a lipoid nature.

It seems, therefore, that the different toxins met with in pellagra have a predilection for certain parts of the nervous system, attacking either the cellular elements or the myelinated tracts. In this case the spinal cord appearances were similar to those seen in the sub-acute combined degeneration of pernicious anaemia ; the pyramidal tracts being especially involved in the lower segments of the cord and the columns of Goll and the spino-cerebellar tracts in the upper.

A. D. B.

HANSEN (P.) Et tilfaelde af sekundaer pellagra hos en mand. [**A Case of Secondary Pellagra in a Man.**]—*Ugeskr. f. Laeger.* 1934. Vol. 96. pp. 943–945. With 3 figs.

The first case of pellagra recognized as such in a man in Denmark is recorded by Hansen who, like many of his Danish colleagues, has been on the look out for secondary pellagra since HESS-THAYSEN published his investigations on the subject.

The patient was a tramway official, aged 44, of a neurasthenic temperament. Having been told in 1905 that he suffered from colitis, and that it required dieting, he took his régime henceforth so much to heart that he dispensed altogether with milk and the coarser forms of bread. His neurasthenia merged into insanity, and he developed the characteristic symmetrical dermatitis, pigmentation, and hyperkeratosis of pellagra. Other manifestations included diarrhoea, indigestion, vomiting, anorexia and considerable loss of weight. No local treatment of the dermatitis was attempted, and a rapid and striking recovery was effected merely by a change of diet. He was given medicinal yeast, dried liver, eggs, oranges and butter, as well as certain preparations supposed to be rich in vitamins. His recovery was the more dramatic as, when this dietetic change was instituted, he seemed to be moribund. *C. Lillingston.*

Rud (Einar). Et tilfaelde af latent pellagra. [**A Case of Latent Pellagra.**]—*Hospitalstidende.* 1934. May 1. Vol. 77. No. 18. pp. 513–516.

This patient had no rash or pigmentation of skin and the diagnosis of pellagra rested on the improvement in response to a dietary rich in vitamin B. *A. G. B.*

Slatineanu (Al.), Nitulescu (J.), Francke (M.), Sibi (M.) & Paraschivescu (Z.). L'ammoniurie et ses rapports avec l'équilibre acidobasique dans la pellagre. [**Ammonuria and Liver and Kidney Function in Pellagra.**]—*C. R. Soc. Biol.* 1934. Vol. 116. No. 26. pp. 1110–1112.

——, ——, Sibi (M.), Cantacuzino (L.) & Lupu (E.). De l'insuffisance hépatique dans la pellagre.—*Ibid.* pp. 1113–1115.

——, ——, ——, Francke (M.) & Veit (E.). Exploration fonctionnelle du rein chez les pellagreux.—*Ibid.* pp. 1115–1117.

Observations upon " ammonuria " and liver and kidney function in pellagra are recorded and described.

1. Observations were made upon 70 pellagrins in whom dermatitis was fully developed. In 57 cases (81 per cent.) the ammoniacal coefficient of the urine was higher than normal. This " ammonuria " often coincides with a condition of acidosis in the blood and may be considered as a reaction on the part of the organism against the acidosis. Various other biochemical estimations are also recorded.

2. Hepatic insufficiency in pellagra has been recognized for a long time. The authors employed various methods for determining liver function, and hepatic insufficiency, by at least one of these tests, was proved to be present in 88 per cent. of pellagra cases.

3. A study of the literature shows that some observers have noted defective kidney function in a certain number of pellagrins. In the present series of experiments various tests were employed, including estimations of blood nitrogen, urea and phenolsulphonepthalein elimination, and chloride concentration capacity of the kidney. Of 55 pellagra cases observed, only 19 gave normal results in response to these tests ; 45 per cent. showed an altered Ambard's coefficient ; 30 per cent. showed a defective elimination of phenolsulphonepthalein ; and 21 per cent. were found to have a defective chloride concentration capacity. Slight albuminuria was frequently found, but this is not regarded as certain evidence of functional renal trouble. *A. D. B.*

Helmer (O. M.), Fouts (Paul J.) & Zerfas (L. G.). **Gastro-Intestinal Studies. III. Determinations of Enzymes on Autopsy Specimens from Cases of Pernicious Anemia and Pellagra.**—*Arch. Intern. Med.* 1934. May. Vol. 53. No. 5. pp. 675–679. [14 refs.]

The authors give their conclusions as follows :—

" 1. The gastric mucosa obtained from three patients with pernicious anaemia contained no pepsin or rennin.

" 2. Pepsin and rennin were present in the gastric mucosa of pellagrins, although in smaller quantities than in persons who died of cardiac failure and pulmonary embolism.

" 3. The pancreatic tissue from the persons with pernicious anemia and pellagra contained normal amounts of tryptic, amylolytic and lipolytic enzymes as compared with that from persons who had died of cardiac failure and pulmonary embolism.

" 4. The duodenal mucosa from persons with pernicious anemia and pellagra produced sufficient enterokinase to activate fully the trypsinogen of the pancreatic tissue." *A. D. B.*

Stannus (Hugh S.). **Vitamin A and Pellagra.** [Correspondence.]—*Lancet.* 1934. Oct. 6. p. 785.

In reference to a leading article on Vitamin A and the skin Dr. Stannus points out that the skin lesions under discussion, now considered to be a manifestation of Vitamin A deficiency, were first described in 1911 by himself in association with pellagra. Other lesions noted were irregular erosions of the tongue, excoriations at the angles of the lips and palpebral fissures, erosions about the prepuce, anus and vulva. Other symptoms such as dimness of vision, apathy, diarrhoea have been associated with these skin lesions—all symptoms common to pellagra. He recalls that Mellanby believes pellagra to be due to a Vitamin A deficiency, and to meet the objections of Goldberger's followers suggests that Vitamin A may be rendered ineffective in the absence of some other nutritional factor such as Vitamin B_2. Stannus thinks that pellagra may easily go unrecognized. *A. G. B.*

Ghaloungui (Paul). Pellagra in Sudanese Millet-Eaters. [Correspondence.]—*Lancet.* 1934. July 21. p. 164.

Hiemcke (H. J. Th.). Pellagroied bij aphthae tropicae.—*Nederl. Tijdschr. v. Geneesk.* 1934. Aug. 25. Vol. 78. No. 34. pp. 3869–3877. With 5 figs. on 4 plates. [21 refs.] French summary (3 lines).

REVIEWS AND NOTICES.

MÖNNIG (H. O.) [B.A., Dr. Phil., B.V.Sc., Professor of Parasitology, Faculty of Veterinary Science, University of Pretoria, etc.]. **Veterinary Helminthology and Entomology. The Diseases of Domesticated Animals caused by Helminth and Arthropod Parasites.**—pp. xvi + 402. With 264 figs. (22 on 12 plates). 1934. London: Baillière, Tindall & Cox, 7 & 8 Henrietta Street, W.C. 2. [30s.]

As the author points out in his preface, no text book which deals adequately with parasitic diseases of the domesticated animals has appeared since NEUMANN'S work in 1892. In the interval, there has been a considerable increase in our knowledge, especially with regard to anthelmintics and methods of control. As a teacher the author has experienced the want of a suitable text book for veterinary students and practitioners, and the present work is an attempt to supply that need. It is no exaggeration to say that Prof. Mönnig has admirably succeeded in his object, and that the present generation of veterinary students and even practitioners owe him a debt of gratitude. It is not encyclopaedic, such is not possible. Certain items which appeal to us in Europe are missing, but the author has tuned in to our atmosphere to a surprising degree, although the African veldt often looms up before us. There is a short account of several aspects of parasitism in general which is followed by a section on technique. This deals with the collection and preservation of worms and arthropods, the making of permanent preparations, and clinical diagnostic methods. Among the latter are included the skin scraping, the direct smear, concentration, floatation and egg counting methods, faecal culture, blood examination and the allergic reactions. It is doubtful whether the student could carry out the Casoni test for echinococcosis from the description on page 21. The section dealing with helminths contains sufficient morphological details to make the classification, life cycles and habits clear and interesting. The most important parasitic diseases are each dealt with in the following manner, the morphology of the parasite, life cycle, pathogenesis, symptoms, post-mortem, diagnosis, treatment and prophylaxis.

There are figures of the eggs of the common trematodes, cestodes, and nematodes. One misses *Limnaea truncatula* in Plate I of the shells of various important snail intermediate hosts of trematodes. In the Plate too, one notices the author uses *Lymnaea* but in the text we see *Limnaea*. Fascioliasis is briefly yet fully discussed in all its aspects. Apart from *Diphyllobothrium* and *Sparganum* the only order of cestodes dealt with is that of the *Cyclophyllidea*, and taeniasis receives full consideration. In the round worms the superfamilies of former days are now natural orders. Ascariasis, haemonchiasis, trichostrongylosis, oesophagostomiasis, respiratory helminthiasis, equine strongylidosis, and many other worm diseases of animals and birds are dealt with.

It is refreshing to see a short account of leeches, many authors being content to dismiss them by saying that they are not regarded as helminths. Among arthropods the following classes are considered, viz: Crustacea, Myriapoda, Pentastomida, Insecta and Arachnida, the latter two receiving most attention. After a general account, the blood-sucking flies, those causing internal and external myiasis,

the lice, the fleas, the ticks, mange mites and pentastomes are briefly but sufficiently dealt with.

There is a host parasite list with page references for each of the species with which the author deals, viz: man, the horse, sheep, goat, ox, pig, dog, cat, fox, rabbit, and several other mammals; among birds, although there are others, the chief are the fowl, turkey, pigeon, duck, goose. *Nematodirus filicollis* is not listed as a parasite of any of the ruminants. The index will not be found complete enough for the practitioner, but perhaps this will be compensated by the host parasite list.

The volume is well printed, illustrated and bound. It should be a guide to the student and should find a place among the reference books of the general practitioner who wants to keep abreast of the times. *A. W. Noel Pillers.*

GIRGES (Rameses). **Schistosomiasis (Bilharziasis).** With a Foreword by Professor Ernest Carroll FAUST.—pp. xii+529. With 1 plate, 185 numbered & unnumbered figs. 1934. London: John Bale, Sons & Danielsson, Ltd., 83–91 Great Titchfield Street, W.1. [25s.]

From his own great experience in Egypt Girges fully describes haematobian and mansonian schistosomiasis and relies mainly on FAUST and MELENEY for what he has to say on the japonic infection. His study of the literature of all forms has been searching, as the number of his citations show, but except for the chapters on Egyptian splenomegaly and the japonic infection there is no list of references; so that for these the reader will be thrown back either on this *Bulletin* to which the author acknowledges invaluable indebtedness, or to KHALIL's Bibliography. The book deals severally and in detail with history, parasitology, epidemiology and geographical distribution, practical study including that of the molluscan hosts, the three infections, specific treatment, and prophylaxis. In the mansonian disease prominence is given to the visceral or splenomegalic type, whose relation to infection by male worms Girges has done so much to clarify. The treatment of all aspects is very thorough. The book is, however, difficult reading with much repetition, and would be greatly improved by drastic reduction. The drawings are excellent; so are the reproductions of photomicrographs by FAUST on the section dealing with the japonic infection, but those of the author would have been improved by lower magnification. Nevertheless this is a fine piece of work written by a busy practitioner and excellently put out. *Clayton Lane.*

LOBEL (L. W. M.). **Lepra bubalorum.**—*Dept. v. Econom. Zaken Nederl.-Indië. Veeartsenijk. Meded. No. 81.* 1934. 234 pp. With 37 figs. on 24 plates.

Apart from leprosy in the human being rat leprosy is the only form of the disease, or of a similar disease, which has hitherto been recognized. Evidence is now forthcoming that the skin affection here described in buffaloes is caused by an acid-fast organism with most of the characters of a leprosy bacillus. Clinically the disease shows itself in the form of a nodular condition of the skin, where the nodules vary from about 5 to 60 mm. in size and may lie in juxtaposition with one another to form large aggregations. These may ulcerate. Altogether in the comparatively short space of time from 1925 to 1934 twenty-one

cases have been discovered in Java and Celebes. The sickness is a very chronic one and indeed the animal is not disabled and is regularly used for work. In two of the cases lesions were present not only in the skin but in the nose. Acid-fast organisms of bacillary type, but also as dark round granules of disintegration, are always found in the skin nodules. They are grouped in typical bundles and globi in very characteristic form, which can be made out with quite low magnifications. These have been found in the nasal lesions as well as the skin, once in regional lymph nodes, but never in the blood or organs. Cultures and attempts to infect other buffaloes, guineapigs, rabbits, pigeons, white rats and mice have all proved negative. The ophthalmic reaction with bovine tuberculin was positive in three out of eight cases. Microscopical sections are troublesome to get owing to the hardness of buffalo skin. The lesion is describable as a granuloma localized in the corium and consisting of highly cellular fatty tissue, containing giant cells, large macrophages (granuloma cells), fibroblasts, a few lymphocytes and still fewer polymorphs : it is not sharply demarcated from the surrounding tissue. Necrosis and calcification are very commonly present. In making a differential diagnosis the field is restricted to diseases due to pathogenic acid-fast bacilli. Tuberculosis and paratuberculosis can be excluded, while the great similarity of grouping of the bacilli, the occurrence of large vacuoles and macrophages (lepra cells) packed with the organisms, and the fatty degeneration combine to form a picture greatly similar to that seen in human leprosy. The conclusion drawn by the author is that " lepra bubalorum is a chronic infectious disease caused by acid fast organisms and that the disease agrees closely in its characters with human leprosy." *W. F. Harvey.*

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Am. signifies Amoebiasis and Amoebic Dysentery.
Bb „ Beriberi and Epidemic Dropsy.
Bl. „ Blackwater.
B.R. „ Book Review.
Chl. „ Cholera.
C.Bu. „ Climatic Bubo and Lymphogranuloma Inguinale.
Der. „ Tropical Dermatology.
Dys. „ Dysentery (Bacillary and Unclassed).
Ent. „ Medical Entomology.
Fev. „ Fevers.
G.V. „ Granuloma Venereum.
Hel. „ Helminthiasis.
Hist. „ Historical.
H.S. „ Heat Stroke.
K.A. „ Kala Azar.
Lep. „ Leprosy.
Lept. signifies Leptospirosis.
Mal. „ Malaria.
Misc. „ Miscellaneous.
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Baer, J., with Joyeux & Houdemer, 406 (Oph.), 782 (Hel.)
——, with —— & Martin, 387 (Hel)
Baffé, O., with Nicolau, Kopciowska & Mathis, 842 (Y.F.)
Baffet, O., with Mathis, 845 (Y.F.)
Bagchi, K. N., 770 (Hel.)
Bailly, J., with Remlinger, 146 *quat.*, 148, 639, 640, 647 *bis* (Rab.)
Baliña, P. L., (13) (Lep.)
—— & Puente, J. J., (266) (Lep.)
Balkovskaya, N., with Jantchur, (726) (Mal.)
Ballif, L., with Ciuca & Chelarescu-Vieru, 687 *bis* (Mal.)
Balliste, R., with Sergent & Manceaux, 87 (R.F.)
Balmus, G., with Nicolau, Kopciowska & Mathis, 843 (Y.F.)
Baltazard, M., with Blanc & Noury, 243, 626 (Fev.)
——, with ——, —— & Barnéoud, 248 (Fev.)
——, with ——, —— & Fischer, 87 (R.F.)
Barbieri, A., 417 (Mal.)
Barbosa, A., (726) (Mal.)
Bargy, M., 403 (Oph.)
Bariéty, M., with Troisier, Erber & Gabriel, 515 (Lept.)
Barling, B., 405 (Oph.)
Barlovatz, A., 205, 206, 556 (S.S.)
Barnéoud, J., with Blanc, Noury & Baltazard, 248 (Fev.)
Barral, R. E. L. & Luc, R. H. L., 14 (Oph.)
Barraud, P. J., (72) (Ent.), 179 (Mal.), 517 (B.R.)
Barrera, B., (361) (Misc.)
Barrett, J. W., 323 (Misc.)
Barrett, R. E., (36) (Pl.), 572 (S.S.)
Barrowman, B., 184, 693 (Mal.)
Bartman, J., 547 (Lep.)
Basilewsky, B. G., 94 (Lept.)
Baumann, H., with Schwetz & Peel, 173 (Mal.)
——, with ——, —— & Belhommet, 410 (Mal.)
——, with ——, —— & Droeshaut, 418, (474) (Mal.)
Bayer, G. & Elbel, H., 102 (Sn.)
Beach, T. D., with Faust, Wells & Adams, 800 (Hel.)
Becker, E. R., 352 (Misc.)
Becker, S. W., 318 (Der.)
Bedell, A. J., 403 (Oph.)
Beeuwkes, H. & Mahaffy, A. F., 829 (Y.F.)
Behrman, S., 786 (Hel.)
Beijnen, G. J. W. K., (435) (Mal.)
Beklemishev, V. N., 468 (Mal.)
Bekman, A., with Nabokoff & Gorjainoff, 57 (Ent.)
Belhommet, with Schwetz, Baumann & Peel, 410 (Mal.)
Bell, P. S., 820 (Misc.)
Bellincioni, (726) (Mal.)
de Benedetti, A., (72) (Ent.), 188, 710 (Mal.)
Benelli, C., Zuccarelli, J. & Sautet, J., 660 (K.A.)

Benyamine, G., (812) (Hel.)
Berberian, D. A., with Yenikomshian, 373 (Hel.)
Beregoff, P., (726) (Mal.)
Bergman, R. A. M., Sitanala & Sardjito, 263 (Lep.)
Bergondi, J., with Meersseman & Friess, 111 (Hel.)
Berland, L., with Mathis, 71 (Ent.)
Bernard, P., 548, 554 *bis* (Lep.)
——, with Afanador, 872 (Lep.)
Bernard, P. N. & Guillerm, J., 43 (Chl.)
——, Raynal, J. & Liang, W., 43 (Chl.)
Bernhoff, F. G., with Kuteischikow & Dosser, 238 (Fev.)
Berntrop, J. C., Jr., 748 (Misc.)
Berny, 4 (Lep.)
Berny, P., 209, 572 (S.S.), 548 (Lep.)
——, with Nodenot, 265 (Lep.)
——, with Tisseuil, 265 (Lep.)
Berthier, J., 387 (Hel.)
Bertrand, I., with Guillain, Mollaret & Lereboullet, 903 (Pel.)
Besredka, A., 105 (Sn.)
Bezemer, F., 93 (Lept.)
Bhalerao, G. D., 780 (Hel.)
Bhatnagar, S. S., with Buist & Carr, 318 (Der.)
Bibliography of Helminthology Year 1931, 143 (B.R.)
Bibliography of Helminthology Year 1932, (812) (Hel.)
Bie, G. S., with Aalsmeer, 19 (Bb.)
Bien, C., (314) (Chl.)
Biggam, A. G. & Ghalioungui, P., 280 (Am.), 489 (Pel.), 798 (Hel.)
——, Hashim, M. & Ghalioungui, P., 382 (Hel.)
Bilfinger, F., with Lépine, 309 (Pl.), 618, 623 (Fev.)
——, with —— & Markianos, 871 (Lep.)
Binford, C. H., 769 (Hel.)
Biologie Médicale, 144 (B.R.), (219) (S.S.)
Bishopp, F. C., Cory, E. N. & Stone, A., (72) (Ent.)
——, with Laake, Parman & Roark, 61 (Ent.)
Bivol, E., with Badensky, 180 (Mal.)
Blacher, W., 516 (R.B.F.)
Blackaby, E. J., 339 (Misc.)
Blacklock, D. B., 269 (Y. & S.)
Blanc, G., Noury, M. & Baltazard, M., 243, 626 (Fev.)
——, ——, —— & Barnéoud, J., 248 (Fev.)
——, ——, —— & Fischer, 87 (R.F.)
Bliss, A. R., Jr. & Gill, J. M., 504 (Y.F.)
Blondin, P. & Riou, M., 340 (Misc.)
Blue, R., 877 (Pl.)
Bobes, S., with Proca & Jonnesco, 642 *bis* (Rab.)
Bock, H., 150 (Rab.)
Bodenheimer, F. S., (189) (Mal.)
Bodet, H., (888) (Pl.)
Boettger, C. R., 471 (Mal.)
Bogliolo, L., 656 (K.A.)
Bohec, J., (36) (Pl.)
Bohnstedt, R. M., with Moncorps, 581 (S.S.)
Bolotina, A., with Tarejew, Epstein, Gantaeva & Raskin, 174 (Mal.)
Boncinelli, U., 250 (Fev.)
—— & Aradas, A., 33 (Pl.)
Bonnal, G., Joyeux, C. & Bosch, P., 120 (Hel.)
Bonnet, P., with Lefrou, 552 *ter*, 867 *bis*, 873 (Lep.)
Borzenkov, A. & Donskov, G., 307 (Pl.)
Bosch, P., with Bonnal & Joyeux, 120 (Hel.)
Bose, A. N., 769 (Hel.)
Bose, J. P., 828 (B.R.)
Bose, S. C., with Chopra, 482 (Bb.)
Bose, S. K., with Ghosh & Gupta, (47) (Chl.)
Boucher, H., 379 (Hel.)
Boughton, D. C., 354 (Misc.)
Bouillat, 870 (Lep.)
Bourguignon, G. C., 593 (S.S.)
——, with Valcke, 169 (Mal.)
Bourret, with Delpy, (603) (S.S.)
Bousfield, C. E., 868 (Lep.)
Bovet, D. & Demanche, L., 175 (Mal.)
Boyd, G. H., 182 (Mal.)
Boyd, M. F., 419 (Mal.)
—— & Stratman-Thomas, W. K., 165, 166, 688, 701 (Mal.)
Boyé, (189) (Mal.), 309 (Pl.), 831, 834 (Y.F.), 876 (Pl.)
Boyers, L. M., (289), (655) (Am.)
Bradley, G. H., 742 (Misc.)
Brahmachari, P., 662 (K.A.)
v. Brand, T., Holtz, F. & Vogel, H., 374 (Hel.)
Bransburg, M., Gorelik, S. & Schichobalowa, N., 782 (Hel.)
Brás de Sá, L. J., (726) (Mal.)
Bratigny, J., 776 (Hel.)
Bredmose, G. V., 900 (Pel.)
Breinl, F., 634 (Fev.)
Bridges, R. F., 632 (Fev.)
Brissy, G., with Jehl, 236 (K.A.)
Bristol, L. M., with Ahmann, 125 (Hel.)
British East African Territories, Conference of Governors of: 731 (Misc.)
British Empire Leprosy Relief Association, (555) (Lep.)
British Medical Journal, 186 (Mal.)
Bromfield, R. J., with Fairley, 176 (Mal.)
Brooks, A. G., with Shortt, 638 (Rab.)
Broom, J. C., with Findlay, 499 (Y.F.)
Brown, H. C., 352 (Misc.)
Brown, J. Y., 167, (189) (Mal.)
Browne, J. A., 780 (Hel.)
Brownfield, O. D., 665 (K.A.)
Brug, S. L., 708 (Mal.)
Brummelkamp, R., 338 *bis*, 349 (Misc.)
Brumpt, E., 62 *bis* (Ent.), 86 *bis*, 509, 852 (R.F.), 240, 249 (Fev.), (361) (Misc.)
——, Duvoir, M. E. & Sainton, J., 787 (Hel.)
de Brun, R., with Harvier & Lafitte, (726) (Mal.)
Brunelli, P., (400) (Hel.)
Brüning, H., 766 (Hel.)
Buchanan, J. C. R., with Connell, 337 (Misc.)
——, with Mackay & Sanderson, 470 (Mal.)
Buckley, J. J. C., 390, 808 (Hel.)
Buding, E., 499 (Y.F.)
de Buen, S., (92) (R.F.), (655) (Am.)
Buist, T. P., Bhatnagar, S. S. & Carr, L. H., 318 (Der.)
Bulletin de l'Office International d'Hygiène Publique, 773 (Hel.), 875 (Pl.), 896 (Chl.)

Bundesen, H. N., Rawlings, I. D. & Fishbein, W. I., (289) (Am.)
Burke, G. T., (289) (Am.)
Burnet, E., 260 (Lep.)
Burnier & Duché, J., 315 (Der.)
Burowa, C. F., with Kassirsky & Umidowa, 474 (Mal.)
Burowa, L., 168 (Mal.)
Burton, A. W., 736 (Misc.)
Busacca, A., 15 (Oph.)
Butler, C. S., 277 (Y. & S.)
Buttiaux, R., 289 (Dys.)
Buttle, G. A. H., Henry, T. A. & Trevan, J. W., 699 (Mal.)
Buxton, P. A. & Mellanby, K., 739 (Misc.)
Byam, W., 336 (Misc.)

C

Cabassu, H., with Giraud, 229 (K.A.)
Cadham, F. T., 130 (Hel.)
Calcutta, 149 (Rab.), 674, 826 (B.R.)
Calcutta Medical Journal, 20 (Bb.)
Calkins, G. N., 363 (B.R.)
Calvano, U., 46 (Chl.)
Cameron, T. W. M., 766 (Hel.)
Caminopetros, J., 657 (K.A.)
Campbell, C. H., 28 (Pel.)
Campbell, H. G. M., with Chopra & Mukherjee, 173 (Mal.)
van Campenhout, 326 (Misc.)
van Campenhout, E., 543 (Lep.)
Cannon, P. R., 706 (Mal.)
Cantacuzène, J., 40 (Chl.)
——, Ottolenghi, D., Swellengrebel, N. H. & Pittaluga, G., 162 (Mal.)
Cantacuzino, L., with Slatineanu, Nitulescu, Sibi & Lupu, 904 (Pel.)
Carcopino, E., with d'Oelsnitz, 232 *bis* (K.A.)
Cardamatis, J. P., 158 (Mal.)
Cardoso, E., with Soper, Penna, Serafim, Jr., Frobisher, Jr. & Pinheiro, 77 (Y.F.)
Carman, J. A., 108 (Hel.)
Carmichael, J., 68 (Ent.)
Carneiro, J. M., with Patto, 641 (Rab.)
Carothers, J. C., 426 (Mal.)
Carothers, J. C. D., with Anderson, 110 (Hel.)
Carr, L. H., with Buist & Bhatnagar, 318 (Der.)
Carrión, A. L. & Koppisch, E., 316 (Der.)
Casini, G., with Lega, (189) (Mal.)
Castaneda, M. R., with Zinsser, 245, 630 *bis*, (Fev.)
Castellani, A., (319) *bis* (Der.)
Castronuovo, G., 336 (Misc.)
Catacouzenos, E., with Patrikios, 150 (Rab.)
Catanei, A., 315 (Der.)
—— & Legroux, C., 316 (Der.)
——, with Sergent, Edm. & Sergent, A., 418, (436), 704 (Mal.)
Cattaneo, D., 402 (Oph.)
Cawston, F. G., 380 *bis*, 774 (Hel.), (823) *bis* (Misc.)
Ceccarelli, D., (435) (Mal.)
Cedercreutz, A., 538 (Lep.)
Celli, A., 220 (B.R.)
Central Co-operative Anti-Malaria Society, Ld., 412 (Mal.)
Chadwick, C. R., with MacHattie & Mills, 114 (Hel.)
Chagas, E., 599 (S.S.)
Chang, H. H., with Struthers, Lin & Ch'en, 662 (K.A.)
Chang, T., with Hu, 804 (Hel.)
Chao, S. S., with Reiner, 212 (S.S.)
Charlet, with Andréoli, 635 (Fev.), 861 (Oph.)
Chassigneux, with Martin & Rouëssé, 689 (Mal.)
Chatterjee, R., 665 (K.A.)
Chatterjee, S. N., with Gupta, 98 (R.B.F.)
Chatterji, N., 404 (Oph.)
Chatterji, S. K., with Asheshov, I. N., Asheshov, I., Khan & Lahiri, 41 (Chl.)
——, with ——, —— & Lahiri, 42 (Chl.)
Chatterji, S. N., 549 (Lep.)
——, with Muir, 549 (Lep.)
Chatterji, S. P., with Muir, 861 (Oph.)
Chaudhuri, R. N., with Chopra, 654 (Dys.)
Chaudhury, S. G., with Chopra, 179 (Mal.)
Chelarescu-Vieru, M., with Ciuca & Ballif, 687 *bis* (Mal.)
Ch'en, J. T., with Struthers, Chang & Lin. 662 (K.A.)
Chen, W. K., 45 (Chl.)
Chéneveau, R., 743 (Misc.)
Cherry, R. O., with Kellaway & Williams, 100 (Sn.)
Chesneau, P. & Nguyen-duy-Ha, 64 (Ent.)
Chesterman, C. C., 205 (S.S.)
Chiang, W. L. & Yang, C. S., (189) (Mal.)
——, with ——, 681 (Mal.)
Chick, H., 22 (Pel.)
Ch'in, K., 785 (Hel.)
Ch'in, K. Y., with Chung, 663 (K.A.)
Chinese Medical Journal, 1 (Lep.), (314) (Chl.)
Chiyuto, S., 5, 866 (Lep.)
Chopra, G. S., with Chopra, R. N. & Gupta, 667 (Misc.)
Chopra, R. N. & Bose, S. C., 482 (Bb.)
—— & Chaudhuri, R. N., 654 (Dys.)
—— & Chaudhury, S. G., 179 (Mal.)
—— & De, P., 822 (Misc.)
——, Ghosh, N. N. & Mukerji, A. K., 768 (Hel.)
—— & Gupta, B. M. D., 172, 700 (Mal.)
——, Gupta, J. C. & Chopra, G. S., 667 (Misc.)
——, —— & Ganguli, S. K., 176 (Mal.)
——, with Maplestone, 374, 375 (Hel.)
——, Mukherjee, B. & Campbell, H. G. M., 173 (Mal.)
——, Mukherjee, S. N. & Sundar Rao, S., 808 (Hel.)
—— & Sen, B., 652 (Am.)
——, Sen, B. & Sen, S., 282 (Am.)
——, Sen, S. & Sen, B., 652 (Am.)
—— & Wahed, A. K. M. A., (726) (Mal.
Chorine, V., 554 (Lep.)
—— & Gillier, R., 180, (435) (Mal.)
——, Guilliny & Montestruc, E., (873) (Lep.)
—— & Koechlin, D., 705 (Mal.)
——, with Marchoux, 89 (R.F.)
—— & Prudhomme, R., 867 (Lep.)
—— & Rodieux, M., 457 (Mal.)
——, with Tisseuil, 554 (Lep.)
Chorley, C. W., 67 (Ent.)
Christensen, B. V. & Lynch, H. J., 375 (Hel.)

Christophers, R., 725 (Mal.)
Christophers, S. R., 74 (B.R.)
—— & Missiroli, A., 158 (Mal.)
——, Sinton, J. A. & Covell, G., 517 (B.R.)
Christopherson, J. B. & Ward, R. O., 776 (Hel.)
Chrzanowski, B. & Mosing, H., 249 (Fev.)
Chu'an-K'uei Hu & Frazier, C. N., 275 (Y. & S.)
Chun, J. W. H., with Wu Lien-Teh, Pollitzer & Wu, 519 (B.R.)
Chung, H., 781 (Hel.)
—— & Ch'in, K. Y., 663 (K.A.)
——, with Lee, 780 (Hel.)
Chworostuchina, M., 882 (Pl.)
Ciaudo, with Giraud & Montus, 231 (K.A.)
Cibils Aguirre, R. & Saubidet, E. J., (289) (Am.)
Cilento, R. W., 320 (Misc.)
Ciuca, M., Ballif, L. & Chelarescu-Vieru, M., 687 *bis* (Mal.)
Ciurea, I., 374 *bis* (Hel.)
Clapham, P. A., 107 (Hel.)
Clarebout, G., with Mouchet, van Hoof, Duren, Fontana, Henry & Henrard, 831 (Y.F.)
Clarke, L. P., with Findlay, 841 (Y.F.)
Cleland, J. B. & Gray, J. H., (361) (Misc.)
Clemmesen, S., 27 *bis* (Pel.)
Cleyndert, P. C. (95) (Lept.)
Cluver, E. H., 771 (Hel.)
Coatney, G. R., 354 (Misc.)
Cochrane, E., (255) (Fev.)
Colas-Belcour, J., with Roubaud, 58 (Ent.), 720 (Mal.)
Cole, H. I., 546 (Lep.)
Coleman, G. E., 507 *bis*, 849 (R.F.)
Collignon, E., 409, 712 (Mal.)
——, with Senevet, 156, (727) (Mal.)
Colvin, C., Paton, J. T. & Dawes, S. R., (13) (Lep.)
Combiesco, C., with Damboviceanu & Soru, 894 (Chl.)
——, with ——, Wisner & Soru, 893 (Chl.)
Combiesco-Popesco, C. & Wisner, B., 43 (Chl.)
Combiesco, D., 625, 626 (Fev.)
—— & Popesco, C., 242 (Fev.)
——, ——, Stamatesco, S. & Angelesco, J., 245 (Fev.)
——, —— & Vantu, 626 (Fev.)
Congo Belge, 735 (Misc.)
Connal, A., 809 (Hel.)
Connell, W. K. & Buchanan, J. C. R., 337 (Misc.)
Constantinesco, V., with Nicolau & Mathis, 145 (Rab.)
Continentino, L., (266) (Lep.)
Cook, S. S., 740 (Misc.)
Cooke, W. E. & McNair, J. S., (726) (Mal.)
Cooley, R. A., 747 (Misc.)
Cordi, J. M. & Otto, G. F., 395 (Hel.)
Corkill, N. L., 99 (Sn.)
Cormack, H. S. & Anderson, L. A. P., 149 (Rab.), 406 (Oph.)
Cornet, with Dodero, 403 (Oph.)
Cornet, E., 14, 17, 401, 407 (Oph.), 388 (Hel.)
——, with Houdemer & Dodero, 388 (Hel.), 406 (Oph.)
Corongotó, V. S., (726) (Mal.)
Corradetti, A., (435), 725 *ter* (Mal.)
Corson, J. F., 201, 215, 582 *bis*, 583 *bis*, 584 (S.S.)
Cort, W. W., with Otto, 789 *bis* (Hel.)
Corti, E., with Kadaner, 92 (Lept.)
Cory, E. N., with Bishopp & Stone, (72) (Ent.)
Costantino, S., 236 (K.A.)
Coste, F., Saenz, A. & Costil, L., 276 (Y. & S.)
Costedoat, 139 (Sp.)
Costil, L., with Coste & Saenz, 276 (Y. & S.)
Cotti, L., (812) (Hel.)
Cottini, G. B., 6 (Lep.)
Courboulès, P. & Mandillon, G. A. L., (823) (Misc.)
Cousin, E., with Sicé & Dantec, 577 (S.S.)
——, with —— & Rivoalen, 203 (S.S.)
Couvy, 37 (Chl.)
Covell, G., with Christophers & Sinton, 517 (B.R.)
Craig, C. F., 460 (Mal.)
Craighead, A. C., with Shortt, Malone & McGuire, 642 (Rab.)
Crawford, P. J., with Soper & Rickard, 836 (Y.F.)
Crawford, S., 651 (Am.)
Crónica Médico-Quirúrgica de la Habana, 814 (Misc.)
Crosfill, J. W. L., 748 (Misc.)
Cross, B., 677 (Mal.)
Cruveilhier, L., with Haguenau & Nicolau, 648 (Rab.)
Cruz, A., 476 (Bb.)
Cruz, A. O., with West, 478 (Bb.)
Cruz, W. O., 136, 797 *bis*, 798 (Hel.)
Cuénod & Nataf, R., 861 (Oph.)
Culbertson, J. T., 585 (S.S.)
Cummins, R. C., 487 (Pel.)
Cunningham, E. R., Kilborn, L. G., Maxwell, J. L., Morse, W. R., Mullett, H. J. & Dickinson, F., 323 (Misc.)
Cunningham, J., Theodore, J. H. & Fraser, A. G. L., 847 (R.F.)
Cushing, E. C. & Patton, W. S., 359 (Misc.)
Cusset, J. & Goldenfoun, Z., 136 (Hel.)

D

Daengsvang, S., with Prommas, 801 (Hel.)
Dalal, N. P., with Greval, 307 (Pl.)
——, with Row & Gollerkeri, 172 (Mal.)
Damany, P., with Plazy, 120 (Hel.), (252) (Fev.)
Damboviceanu, A., 46 (Chl.)
——, Combiesco, C. & Soru, E., 894 (Chl.)
——, ——, Wisner, B. & Soru, 893 (Chl.)
Dang, M. L., with Still, (655) (Am.)
Dantec, with Sicé & Cousin, 577 (S.S.)
Das, G., with Gupta & Majumdar, 411 (Mal.)
Daspect, M. & Jonchères, H., 284 (Am.)
Dau, H., with Pijper, 241 (Fev.)
David, J., (726) (Mal.)
David, J. C., Rajamanikam, N. & Krishnaswamy, R., 821 (Misc.)
David, N. A., Johnstone, H. G., Reed, A. C. & Leake, C. D., 283 (Am.)
Davis, G. E. & Parker, R. R., 249, 633 (Fev.)
——, —— & Walker, M. E., 634 (Fev.)
Davis, M. E., with Macht, 102 (Sn.)

Davis, N. C., 80, 497, 839, 841 (Y.F.)
——, Frobisher, M., Jr. & Lloyd, W., 81 (Y.F.)
Dawes, S. R., with Colvin & Paton, (13) (Lep.)
Day, P. L., 860 (Oph.)
De, M. N., 663 (K.A.)
De, P., with Chopra, 822 (Misc.)
Deasis, C., 747 (Misc.)
Decourt, P., 693 (Mal.)
——, with Sicault, 431, 432 (Mal.)
Dejou, L., with Huard & Roques, (655) (Am.)
De La Camara Cailhau, P., (726) (Mal.)
Delanoë, E., 15 (Oph.), (555) (Lep.)
Delanoë, P., 506 *bis*, 507 (R.F.)
Delbove, with Ragiot, (96), 854 (Lept.)
Delpy & Bourret, (603) (S.S.)
Demanche, L., with Bovet, 175 (Mal.)
Demidowa, L. W., with Kritschewski, 181 (Mal.)
Demina, N., 279 (Am.)
Demurtas, M. P., (435) (Mal.)
Denney, O. E., 257, 539 (Lep.)
—— & Eddy, B. E., 9 (Lep.)
Denny-Brown, D., 786 (Hel.)
Deschiens, R., 284 (Am.)
Des Essarts, J. Q., 286 (Dys.), 620 (Fev.)
—— & Lefrou, G., 550, 867 (Lep.)
Dévé, F., 121, 387, 785 *bis* (Hel.)
DeWolf, H. F., with Spies, 23 (Pel.)
Dhont, C. M., Jr., 705 (Mal.)
Diamantis, A., 775 (Hel.)
Dias, E., 597 (S.S.)
Dickinson, F., with Cunningham, Kilborn, Maxwell, Morse & Mullett, 323 (Misc.)
Dinger, J. E., (95) (Lept.)
Dixon, H. B. F., 119 (Hel.)
—— & Smithers, D. W., 786 (Hel.)
Dixon, P. K., 16 (Oph.), 379 (Hel.)
Dmitriev, I. D. & Artemiev, A. I., 57 (Ent.)
Do Amaral, A., 104 *bis* (Sn.)
—— & Monteiro, J. L., 616 (Fev.)
Dobell, C., 352 (Misc.)
Dodero & Cornet, 403 (Oph.)
——, with Houdemer & Cornet, 388 (Hel.), 406 (Oph.)
Dodero, J., with Genevray, 646 (Rab.)
Dogra, J. B., with Lal, 235 (K.A.)
Donatien, A., with Parrot & Lestoquard, 229 (K.A.)
——, with Sergent, E., Sergent Et. & Parrot, 423 (Mal.)
——, with ——, ——, —— & Lestoquard, 226 (K.A.)
Donskov, G., with Borzenkov, 307 (Pl.)
Dosser, E. M., with Kuteischikow & Bernhoff, 238 (Fev.)
Dossère, E. M., with Silber, 623 *bis* (Fev.)
Dostrowsky, A., 666 (K.A.)
Dove, W. E., with Hull & Prince, 745 (Misc.)
Downes-Shaw, A., 542 (Lep.)
Drake, A. A., with Rice & Smith, 858 (Oph.)
Dressler, I., with Manteufel, 92 (R.F.)
Dreyfuss, A., (435) (Mal.)
Drinker, C. K., Field, M. E. & Homans, J., 806 (Hel.)
——, Wislocki, G. B. & Field, M. E., 133 (Hel.)
Droeshaut, with Schwetz, Baumann & Peel, 418, (474) (Mal.)
Duché, J., with Burnier, 315 (Der.)
Dudley, S. F., 493 (Y.F.), 787 (Hel.)
Duff, D., 496 (Y.F.)
Dujardin-Beaumetz, E., (36) (Pl.)
Duke, H. L., 196, 564, 565, 566 (S.S.)
Dumont, P., with Van den Branden, 578 (S.S.)
Dumont, R., (290) (Am.)
Duncarf, D., (435) (Mal.)
Dunn, L. H., 63, 70 (Ent.), 218 (S.S.)
Dunnewold, R., (823) (Misc.)
Dupont, R., (812) (Hel.)
Durand, R., with Laigret, 246, 623 (Fev.)
Duren, A., with Mouchet, van Hoof, Fontana, Clarebout, Henry & Henrard, 831 (Y.F.)
Durieux, C., with Mathis, 505, 849 (R.F.)
——, with —— & Advier, 87, 505 (R.F.)
Dusan, J., with Vigne (555) (Lep.)
Duval, C. W. & Holt, R. A., 552, 871 (Lep.)
Duvoir, M. E., with Brumpt & Sainton, 787 (Hel.)
Dyer, R. E., 622 (Fev.)

E

Earle, W. C., 681 (Mal.)
East African Medical Journal, 660 (K.A.)
Eaton, L. M. & Feinberg, S. M., 165 (Mal.)
Eckhardt, A. E., 169 (Mal.)
Eddy, B. E., with Denney, 9 (Lep.)
Edgecombe, W., 336 (Misc.)
Edwards, F. W., 55 (Ent.)
Eerkens, J. W., with de Langen, 125 (Hel.)
Egorov, A., 305 (Pl.)
Egypt, 18, 862 (Oph.), 485 (Pel.), 764 (Hel.)
Eisma, M., 392 (Hel.)
Ejercito, A., (726) (Mal.)
Elbel, H., with Bayer, 102 (Sn.)
Eldin, M. S. & Hassan, A., 767 (Hel.)
El Diwany, M. A. M., 775 (Hel.)
Eliot, M. M. & Jackson, E. B., 346 (Misc.)
El Khalidi, H. F., 683 (Mal.)
Elliot, R. H., 404, 860 (Oph.)
Elmes, B. G. T., 317 (Der.)
——, with Smith, 274 (Y. & S.)
Elsbach, L., 132 (Hel.)
Elshout, J. M. & Lentjes, L. J. M., 477 (Bb.), 603 (S.S.)
Emerson, G. A., Anderson, H. H. & Leaek, C. D., 262 *bis*, 263, 868 (Lep.)
——, with Anderson & Fisher, 869 (Lep.)
—— & Salle, A. J., 547 (Lep.)
Emile-Weil, P., 690 (Mal.)
Epstein, B., 771 (Hel.)
Epstein, E., with Tareev & Gontaewa, 703 (Mal.)
——, with ——, Pikul, Gontaeva & Raskin, 698 (Mal.)
Epstein, G. V., 278 (Am.)
—— & Zertchaninov, L., 279 (Am.)
Epstein, H., with Tarejew, Gantaeva, Raskin & Bolotina, 174 (Mal.)
Erber, B., with Troisier, Bariéty & Gabriel, 515 (Lept.)
Erfan, M., 114 (Hel.)
Ernstene, A. C. & Riseman, J. E. F., 237 (Fev.)
Erspamer, V., 775 (Hel.)
Escalar, G., 686 (Mal.)
——, with Pecori, 186 (Mal.)

Eschbach, H., 251 (Fev.)
Ess, B. J., 262 (Lep.)
Essed, W. F. R., 221 (B.R.)
—— & van Thiel, P. H., 792 (Hel.)
Estrade, F., 879, 884, (888) *bis* (Pl.)
——, with Girard, 879 (Pl.)
Evans, A. M., 723 (Mal.)
Evseeva, V. & Firsov, I., 31 (Pl.)
Ewing, H. E., (72) (Ent.)

F

Fabiani, G., (36) (Pl.)
Fabry, (361) (Misc.)
Faddeeva, T., 32 (Pl.)
Faget, G. H., 164 (Mal.)
Fairbairn, H., 559 (S.S.)
Fairbrother, R. W., 604 (B.R.)
Fairley, N. H. & Bromfield, R. J., 176 (Mal.)
Fakhry, A., 768 (Hel.)
Falta, M., 859 (Oph.)
Farran-Ridge, C. & Lush, D., 285 (Dys.)
Faust, E. C., 129, 381, 776 (Hel.), 335 (Misc.)
——, with Hoffman, 777 (Hel.)
——, Wells, J. W., Adams, C. & Beach, T. D., 800 (Hel.)
Federated Malay States, 153 (Mal.), 728 (Misc.)
Feinberg, S. M., with Eaton, 165 (Mal.)
Fejgin, B. & Sparrow, H., 624 (Fev.)
Feldt, A., 511 (R.F.)
Feng, C. T., 870 (Lep.)
Feng. L., 741 (Misc.), 805 (Hel.)
—— & Hoeppli, R., 746 (Misc.)
——, with ——, 787 (Hel.)
Fennel, E. A., 621 (Fev.)
Fenton, F. G., with Lyle, 862 (Oph.)
Fermi, C., 53 (Ent.)
Fernandez, F., 767 (Hel.)
Fernando, A. S., 858 (Oph.)
Fernan-Nunez, M., (655) (Am.)
Féron, 262 *bis* (Lep.)
Feron, J., 549 (Lep.)
Ferré, 7 (Lep.)
Ferreira, J. C., with Mendes, 657 (K.A.)
Fidanza, E. P., (266) *bis* (Lep.)
Field, M. E., with Drinker & Homans, 806 (Hel.)
——, with —— & Wislocki, 133 (Hel.)
de Figueiredo, J. M. P., 883 (Pl.)
Filatov, A., with Ackerman, 459 (Mal.)
Filho, M. C., (483) (Bb.)
Findlay, G. M., 498, 500, 501 (Y.F.)
—— & Broom, J. C., 499 (Y.F.)
—— & Clarke, L. P., 841 (Y.F.)
Findlay, L., 814 (Misc.)
Fine, A., with d'Aunoy, 148 (Rab.), 630 (Fev.)
Firsov, I., with Evseeva, 31 (Pl.)
Fischer, with Blanc, Noury & Baltazard, 87 (R.F.)
Fischer, O., 336 (Misc.)
Fischl, V., Kotrba, J. & Singer, E., 510 (R.F.)
—— & Schlossberger, H., 518 (B.R.)
——, with Singer, 510 (R.F.)
—— & Singer, E., 851 (R.F.)
——, with —— & Kotrba, 851 (R.F.)
Fishbein, W. I., with Bundesen & Rawlings, (289) (Am.)
Fisher, A. C., 775 (Hel.)
Fisher, B. H., with Anderson & Emerson, 869 (Lep.)
Fishl, M., 648 (Rab.)
Fitzgerald, G. H., 282 (Am.)
Fitzgerald-Moore, D. G., 820 (Misc.)
Fitzwilliams, D. C. L., 803 (Hel.)
Fleck, L., with Hescheles, 621 (Fev.)
Fletcher, W., 631 (Fev.)
Flinker, R., 492, 901 (Pel.)
Flu, P. C., 34 *bis*, (310) (Pl.), 44, 898 (Chl.)
Fontana, L., with Mouchet, van Hoof, Duren, Clarebout, Henry & Henrard, 831 (Y.F.)
Forkner, C. E. & Zia, L. S., 656 (K.A.)
Fosbery, F. W. A., (400) (Hel.)
Foshay, L., 348 (Misc.)
Foster, J. H., (655) (Am.)
Fourie, L., 183 (Mal.)
Fournials, 818 (Misc.)
Fouts, P. J., with Helmer & Zerfas, 905 (Pel.)
Fox, H., 538 (Lep.)
Franchini, G., 850 (R.F.)
Francke, M., with Slatineanu, Nitulescu, Sibi, & Paraschivescu, 904 (Pel.)
——, with ——, ——, —— & Veit, 904 (Pel.)
Franklin, E. M., 427 (Mal.)
Fränkmann, E., with Jelin, 247 (Fev.)
Fraser, A. G. L., with Cunningham & Theodore, 847 (R.F.)
Fraser, S. N. D., 1 (Lep.)
Frazier, C. N., with Chu'an-K'uei Hu, 275 (Y. & S.)
——, with Ngai, 651 (Am.)
Frégonneau, W., 380 (Hel.)
Freund, L., 373 (Hel.)
Fridie, E., 773 (Hel.)
Friedlander, R. D., 802 (Hel.)
Friess, E., with Meersseman & Bergondi, 111 (Hel.)
Frobisher, M., Jr., 83 (Y.F.)
——, with Davis & Lloyd, 81 (Y.F.)
——, with Soper, Penna, Cardoso, Serafim, Jr. & Pinheiro, 77 (Y.F.)
Fróes, H., (189) (Mal.), (823) *bis*, (824) *bis* (Misc.)
Froes, H. P., (813) *bis* (Hel.), (726) (Mal.)
Frye, W. W. & Meleney, H. E., 284 (Am.)
Funayama, J.-I., 647 (Rab.)
Futran, G. S., with Prendel & Zagorovskii, 471 (Mal.)

G

Gabriel, P., with Troisier, Bariéty & Erber, 515 (Lept.)
Gaehtgens, with Peter, 819 (Misc.)
Gaffney, H. J. O'D. B., (361) (Misc.)
Gaiskaya, E. L., 279 (Am.)
Gajdos, E. & Tchang, J., 629 (Fev.)
Galante, E., 860 (Oph.)
Galliard, H., 254 (Fev.), 745 (Misc.)
—— & Sautet, J., 722 (Mal.)
Galli-Valerio, B., 107 (Hel.)
Galloway, I. A., 638 (Rab.)
Ganguli, S. K., with Chopra & Gupta, 176 (Mal.)

Gantaeva, A., with Tarejew, Epstein, Raskin & Bolotina, 174 (Mal.)
Garcia, A., with de Leon & de Jesus, 350 (Misc.)
Garcia, O., 274 *bis* (Y. & S.)
Garcia Corselas, T., with Mezquita Lopez, (400) (Hel.)
Garrison, H. F., 393 (Hel.)
Gaspare, L., 850 (R. F.)
Gater, B. A. R., 58 (Ent.), 678 (Mal.), 755 (B.R.)
Gaud, M., 306 (Pl.)
Gauducheau, A., (483) (Bb.)
Genevray, J. & Dodero, J., 646 (Rab.)
Geoghegan, A. J., 217 (S.S.)
George, P. V. & Webster, W. J., 878 (Pl.)
Gerbinis, P., 265 (Lep.), 458 (Mal.)
Germain, L., (824) (Misc.)
Getzowa, S., Stuart, G. & Krikorian, K. S., 150 (Rab.)
Ghalioungui, P., (905) (Pel.)
——, with Biggam, 280 (Am.), 489 (Pel.), 798 (Hel.)
——, with —— & Hashim, 382 (Hel.)
Ghosh, B. N., 706 (Mal.)
Ghosh, H., Gupta, B. K. S. & Bose, S. K., (47) (Chl.)
Ghosh, J. L., with Roy & Nandi, 20 (Bb.)
Ghosh, N. N., with Chopra & Mukerji, 768 (Hel.)
Giamarchi, P. & Sautet, J., 360 (Misc.)
Gibbins, E. G., 56, 57, 60 (Ent.), 460 (Mal.), 812 (Hel.)
—— & Loewenthal, L. J. A., 399 (Hel.)
Gibson, C. R., with Stannus, 484 (Pel.)
Gibson, P. L. & O'Flynn, J. A., 660 (K. A.)
Giemsa, G. & Oesterlin, M., 174 (Mal.)
Giglioli, G., 166 (Mal.)
Gilbert, A. P. W., with Soesilo, 474, 713 (Mal.)
Gilkes, H. A., 335, 751 (Misc.)
Gill, C. A., 424 (Mal.)
Gill, J. M., with Bliss, 504 (Y.F.)
Gillett, J. D., with Wigglesworth, 746 (Misc.)
Gillier, R., with Chorine, 180, (435) (Mal.)
—— & Tisseuil, J., 555 (Lep.)
——, with ——, 265 (Lep.)
Gilly, C., 328 (Misc.)
Ginsburg, J. M., 711 (Mal.), 741 (Misc.)
Giordano, A., 664 (K.A.)
Giovanardi, A. & Mondolfo, U., 765 (Hel.)
Giovannola, A., 455 (Mal.)
——, with Kikuth, 175 (Mal.)
Girard, 7 (Lep.)
Girard, G., 884 (Pl.)
——, with Bablet, 307, 885 (Pl.)
—— & Estrade, F., 879 (Pl.)
——, with Nattan-Larrier, 276 (Y. & S.)
—— & Robic, J., 885 (Pl.)
—— & Woltz, H., 7 (Lep.)
Giraud, P. & Cabassu, H., 229 (K.A.)
——, Mollaret & Monges, F., 660 (K.A.)
——, Montus & Ciaudo, 231 (K.A.)
Girges, R., 767, (813), 790 *bis*, 791 (Hel.), 907 (B.R.)
Giroud, P., with Nicolle, 240 (Fev.)
——, with —— & Laigret, 244 (Fev.)
——, with —— & Sparrow, 628, 629 (Fev.)
Giunta, G., (277) (Y. & S.)
Glusman, M. P., Ssolowiewa, J. V. & Predteschenskaia, L. A., 151 (Rab.)
Goez, Y., 514 (Lept.)
Gohar, M. A., 890 (Chl.)
Goiran, 480 (Bb.)
——, with Toullec, (483) (Bb.)
Golden, R. & O'Connor, F. W., 399 (Hel.)
Goldenfoun, Z., with Cusset, 136 (Hel.)
Gollerkeri, G. V., with Row & Dalal, 172 (Mal.)
Golzowa, R. D., with Popow, 49 (Ent.)
Gomes, J. M., (266) (Lep.)
—— & Antunes, P. C. de A., 872 (Lep.)
Gomes, L. de S., (252) (Fev.)
Gontaeva, A., with Tareev, Epstein, Pikul & Raskin, 698 (Mal.)
Gontaewa, A., with —— & ——, 703 (Mal.)
Gontier & Popoff, 513 (R.F.)
González, H. D., 648 (Rab.)
Goodale, R. H., 397 (Hel.)
Gopsill, W. L., 376, 382 (Hel.)
Gordon, H., 131 (Hel.)
Gordon, H. L., 16 (Oph.)
Goré, R. N., 187 (Mal.)
Gorelik, S., with Bransburg & Schichobalowa, 782 (Hel.)
Gorjainoff, A., with Nabokoff & Bekman, 57 (Ent.)
Gosio, R., (436) (Mal.)
Gouget, R., 409 (Mal.)
Govi, L., (824) (Misc.)
Goyanes Alvarez, J., 135 (Hel.)
de Graaf, J. F. H. & Levie, L. H., 261 (Lep.)
Gradwohl, R. B. H., (655) (Am.)
Graham-Yooll, M. A., (400) (Hel.)
Gramberg, K. P. C. A., (555) (Lep.)
Grant, A. M. B., 124 (Hel.)
Grantham-Hill, C., 62 (Ent.)
Grasset, E., 104 *bis* (Sn.), (824) (Misc.)
—— & Zoutendyk, A., 103 *bis*, 104 (Sn.)
Gray, J. H., with Cleland, (361) (Misc.)
Green, R., 695 (Mal.)
Greig, E. D. W., 690 (Mal.)
Grenci, E., 237 (Fev.)
Greval, S. D. S. & Dalal, N. P., 307 (Pl.)
Grikurow, W., 882 (Pl.)
Grillo, J., (873) (Lep.)
Grimaldi, V., (824) (Misc.)
Grimard-Richard, L., with Nattan-Larrier, 230, 657 (K.A.)
Grisolia y Juristo, M., (400) (Hel.)
Grizaud, 543 (Lep.)
Grünfeld, A. A., with Serebrennaja & Neumann, 243 (Fev.)
——, Serebrjannaja, A. I. & Neumann, M. W., 622 (Fev.)
Guardabassi, M., 460 (Mal.)
Guillain, G., Bertrand, I., Mollaret, P. & Lereboullet, J., 903 (Pel.)
Guillerm, J., with Bernard, 43 (Chl.)
Guilliny, with Heckenroth, (400) (Hel.)
Guilliny, R., with Chorine & Montestruc, (873) (Lep.)
—— & Montastruc, E., 13 (Lep.)
Gunewardene, T. H., 654 (Dys.)
Gupta, B. K. S., with Ghosh & Bose, (47) (Chl.)
Gupta, B. M., with Sarkar, 21 (Bb.)

Gupta, B. M. D. & Chatterjee, S. N., 98 (R.B.F.)
——, with Chopra, 172, 700 (Mal.)
——, with Wats, 463 (Mal.)
Gupta, C. R. D., with Napier, 661 (K.A.)
——, with —— & Smith, 227 (K.A.)
Gupta, J. C., with Chopra, R. N. & Chopra, G. S., 667 (Misc.)
——, with Chopra & Ganguli, 176 (Mal.)
Gupta, P., Das, G. & Majumdar, N. R., 411 (Mal.)
Gupta, R. C. D., 640 (Rab.)
Gupta, S. K., with Pasricha & de Monte, 42 (Chl.)
Guy, R., (726) (Mal.)
—— & Tran-Van-Nguu, 14 (Oph.)

H

Haagen, E., 84, 844, 846 (Y.F.)
Hackett, L. W., 471 (Mal.)
Hae, L. E., 14, 17 (Oph.)
Haguenau, J., Cruveilhier, L. & Nicolau, S., 648 (Rab.)
Hai, with Mickaniewski, 690 (Mal.)
Hakki, I., 684 (Mal.)
Halber, W., with Hirszfeld, (252) (Fev.)
Hamlyn-Harris, R., 51 (Ent.)
Hanón, J. L., (874) (Lep.)
Hansen, P., 903 (Pel.)
Harley, G. W., (137) (Hel.), 271, 272 (Y. & S.)
Harris, L. J., 28, (492) (Pel.)
Harris, R. H. T. P. & Whitnall, A. B. M., 601 (S.S.)
Harrison, R. W., with Julianelle, 401 (Oph.)
Harrower, G., 390 (Hel.)
Hart, R. W. & Pelikan, E. R., 887 (Pl.)
Harvey, W. H., 744 (Misc.)
Harvier, P., de Brun, R. & Lafitte, A., (726) (Mal.)
Hase, A., 60, 69 (Ent.)
Hassan, A., with Eldin, 767 (Hel.)
Hasselmann, C. M., 1 (Lep.), (189) (Mal.), 269, 273, 275 (Y. & S.)
Hashim, M., 379 (Hel.)
——, with Biggam & Ghalioungui, 382 (Hel.)
van Hasselt, E. H., (361) (Misc.)
Havens, L. C. & Mayfield, C. R., 147 (Rab.)
Hawking, F., 581 (S.S.)
Hayashi, F., 12 (Lep.)
Hayashi, N., 252 (Fev.)
Hayden, R., 430 (Mal.)
Hazato, H., with Nagayo, Mitamura & Tamiya, 242 (Fev.)
Headlee, W. H., 106 (Hel.)
Heagerty, J. J., 539 (Lep.)
Health, Canberra, 320 (Misc.)
Heatly-Spencer, J., 649 (Am.)
Hecht, G., 171 (Mal.)
Hecht, O., 357 (Misc.), 464 (Mal.)
Heckenroth & Guilliny, (400) (Hel.)
Hegner, R., 287, 288 *bis* (Dys.), 649 (Am.)
Heiberg, B., 898 (Chl.)
Heimberger, L. F., 1 (Lep.)
Hellsten, H., 396 (Hel.)
Helmer, O. M., Fouts, P. J. & Zerfas, L. G., 905 (Pel.)
Helmy, M., 383 (Hel.)
Helmy, M. M., with Tomb, 109 (Hel.)
Helpern, M., 689 (Mal.)
Henderson, L. H., (436) (Mal.)
Henrard, C. & van Hoof, L., 184 (Mal.)
——, with ——, 562, 596 (S.S.)
——, with Mouchet, van Hoof, Duren, Fontana, Clarebout & Henry, 831 (Y.F.)
Henry, (436) (Mal.)
Henry, A. F. X., 706 (Mal.)
Henry, E., with Mouchet, van Hoof, Duren, Fontana, Clarebout & Henrard, 831 (Y.F.)
Henry, T. A., with Buttle & Trevan, 699 (Mal.)
Henry, X., 456 (Mal.)
Herbert, H., 405 (Oph.)
Hermans, E. H., 291 (B.R.)
Hertzig, A., with Weigl, 244 (Fev.)
Hescheles, I. & Fleck, L., 621 (Fev.)
Hewer, T. F., 833 (Y.F.)
Heyman, A. M., with Soule, 285 (Dys.)
Hiemcke, H. J. T., (905) (Pel.)
Higgins, M. E., 256 (Lep.)
Hilferty, M. M., with Shattuck, 192 (H.S.)
Hill, R. B. & Tejera, E., 380 (Hel.)
Himmelsbach, C. K., 419 (Mal.)
Himmelweit, F., 90 (R.F.)
Hindle, E., 497 (Y.F.), 512 (R.F.)
—— & White, P. B., 512 (R.F.)
Hinman, E. H., 50, 51, (72) (Ent.), 742 (Misc.)
——, with Kampmeier, (824) (Misc.)
Hirai, T., with Kimura, Misugi & Miyazawa, 251 (Fev.)
Hirano, H., with Schöbl, Vazquez-Colet, Ramirez & Arima, 96 (R.B.F.)
Hiroki, H., 88 (R.F.), (888) (Pl.)
——, with Toda, 510 (R.F.)
Hirszfeld, L. & Halber, W., (252) (Fev.)
Hissette, J., 135 (Hel.)
Hodgman, J. H., 793 (Hel.)
Hoeft, G. L., with Tonney & Spector, (290) (Am.)
Hoeppli, R., 770 (Hel.)
—— & Feng, L. C., 787 (Hel.)
——, with ——, 746 (Misc.)
—— & Watt, J. Y. C., 745 (Misc.)
Hoffman, F. L., 28 *bis* (Pel.)
Hoffman, W. A., 776 (Hel.)
—— & Faust, E. C., 777 (Hel.)
——, with Molina & Rojas, (813) (Hel.)
——, with Pons, 381 (Hel.)
——, —— & Janer, J. L., 778 (Hel.)
Hoffmann, W. H., 3, 553 (Lep.), (85) (Y.F.)
Hofman-Bang, A., 488 (Pel.)
Hogue, M. J., 353 (Misc.)
Holden, H. F., 102 (Sn.)
——, with Kellaway, 99 (Sn.)
Holm, S., (492) (Pel.)
Holmes, F., 786 (Hel.)
Holsendorf, B. E., 309 (Pl.)
Holt, R. A., 552 *ter* (Lep.)
——, with Duval, 552, 871 (Lep.)
Holtz, F., with v. Brand & Vogel, 374 (Hel.)
Homans, J., with Drinker & Field, 806 (Hel.)
van Hoof, L., 210, 579, 588 (S.S.), 377, 811 (Hel.)
—— & Henrard, C., 562, 596 (S.S.)
——, with ——, 184 (Mal.)
——, with Mouchet, Duren, Fontana, Clarebout, Henry & Henrard, 831 (Y.F.)

van Hoof, M. T., with Rodhain, 377 (Hel.)
d'Hooghe, M., 810 (Hel.)
Hoops, A. L., 678, 695 (Mal.)
Horrenberger, R., 87 (R.F.)
Hoshizaki, S., 243 (Fev.)
Hoskins, M., 840 (Y.F.)
Hosono, S., with Nishibe & Miyazawa, 251 (Fev.)
Houdemer, E., 53 (Ent.), 377 (Hel.)
——, Dodero & Cornet, 388 (Hel.), 406 (Oph.)
——, with Joyeux & Baer, 406 (Oph.), 782 (Hel.)
Hsü, H. F., (137), 788 (Hel.)
—— & Watt, J. Y. C., 812 (Hel.)
Hsü-Hsi-fan, 135 (Hel.)
Hu, C. H., 663 (K.A.)
Hu, S. M. K. & Chang, T., 804 (Hel.)
—— & Yen, C., 804 (Hel.)
Huai-Chieh, K. & Yung Tsung, Y., 780 (Hel.)
Huang, K. K., with Yang, 476 (Bb.)
Huard, P., (655) (Am.)
——, Roques, P. & Dejou, L., (655) (Am.)
Huchon, (361) (Misc.)
Huff, C. G., 464 (Mal.)
Hughes, T. A., 341 (Misc.)
Hughes, T. P., 83 (Y.F.)
Huisman, R. G. J. P. & Müller, H., 340 (Misc.)
Hull, J. B., Dove, W. E. & Prince, F. M., 745 (Misc.)
Hulse, C. R., with O'Connor, 133 (Hel.)
Hulshoff, A. A., 344 *bis* (Misc.)
Humann, 327 (Misc.)
Hunt, D. J., with Wheeler, 26, 902 (Pel.)
Huri, M., 311 (Chl.)
Hurst, E. W., 146 (Rab.)
Husamettin, 684 (Mal.)

I

von Ihering, R., (361) (Misc.)
Iimura, Y., 306 (Pl.)
Iio, 648 (Rab.)
Ikeda, K., (655) (Am.)
Ildrim, D., 315 (Der.)
Imagawa, Y., with Kawamura & Itô, 251 (Fev.)
Impastato, A., (824) (Misc.)
Inda, F. F., Vivoli, D. & Vaccarezza, A. J., 659 (K. A.)
Index Veterinarius, 143 (B.R.)
India, 733 (Misc.)
Indian Medical Gazette, (266) (Lep.), 692 (Mal.)
Ingham, T. R., with Lund, 281 (Am.)
Ingram, W. W. & Musgrave, A., 71 (Ent.)
Inouye, K., with Yamada, 799 (Hel.)
International Journal of Leprosy, 538 (Lep.)
Ionesco-Miahaiesti, C., Zotta, G., Radacovici, E. & Badenski, G., 707 (Mal.)
Isabolinski, M. P., Sobolewa, R. M., Stratanowitsch, N. J., Riwkina, S. L. & Moskalewa, T. A., 619 (Fev.)
Ishii, Y., 768 (Hel.)
Ishioka, H., 704 (Mal.)
——, with Morishita & Miyahara, (190) *ter* (Mal.)
——, with —— & ——, 694 (Mal.)
Ishizu, S., 546 (Lep.)
v. Issekutz, B., 211 (S.S.)
Iswariah, V., 100 (Sn.)
—— & Kutumbiah, P., 479 (Bb.)
—— & Nair, V. G., 551 (Lep.)
Itô, T., with Kawamura & Imagawa, 251 (Fev.)
Iwase, Y., 101, 102 *bis* (Sn.)
Iwata, S., 783 (Hel.)
Iyengar, M. O. T., 679 (Mal.), 887 (Pl.)
Iyer, P. V. S., 30 (Pl.)
——, with King, 30 (Pl.)
——, with Pandit & Menon, 33 (Pl.)

J

Jackson, C. H. N., 216 *bis*, 600, 601 (S.S.)
Jackson, E. B., with Eliot, 346 (Misc.)
Jacobson, J., 15 (Oph.)
Jafar, M. & Singh, B., 255 (Fev.)
Jahnel, F., 513 (R.F.)
James, S. P., 171 (Mal.), 832 (Y.F.)
Janer, J. L., with Hoffman & Pons, 778 (Hel.)
Janisch, E., 48, 68 (Ent.)
Jantchur, N. M. & Balkovskaya, N., (726) (Mal.)
Javett, S. N., 738 (Misc.)
Jeanselme, E., 545 (Lep.), 754 (B.R.)
Jehl, E. P. & Brissy, G., 236 (K.A.)
Jelin, W. & Fränkmann, E., 247 (Fev.)
Jellison, W. L., 634 (Fev.)
—— & Philip, C. B., 69 (Ent.)
Jensen, E. V., 488 (Pel.)
Jepps, M. W., 111 (Hel.)
Jepson, F. P., 139 (Sp.)
Jerace, F., (436) (Mal.)
de Jesus, P. I. & de Leon, W., 350 (Misc.)
——, —— & Anzures, P., 350 (Misc.)
——, with —— & Garcia, 350 (Misc.)
——, —— & Ramos, J. M., 350 (Misc.)
de Jesus , Z., 749 (Misc.)
Jettmar, H. M., (888) (Pl.)
Johnson, C. M., with Andrews & Schwartz, 283 (Am.)
Johnson, F. H., (36) (Pl.)
Johnson, P. D., 427 (Mal.)
Johnston, J. M., with Stockman, 23 (Pel.)
Johnstone, H. G., with David, Reed & Leake, 283 (Am.)
——, with Reed, 649 (Am.)
Johnstone, I. L., (726) (Mal.)
Jonchère, 687 (Mal.)
Jonchères, H., with Daspect, 284 (Am.)
Jones, C. A., 776 (Hel.)
Jones, C. M., with Urmy, Ragle & Allen, 481 (Bb.)
Jones, R. F., with Poindexter, 804 (Hel.)
de Jong, R. de J., 550 (Lep.)
Jonnesco, D., 147 (Rab.)
——, with Proco & Bobes, 642 *bis* (Rab.)
Jordan, E. O. & McBroom, J., 669 (Misc.)
Jordan, P., 866 (Lep.)
Jorge, R., 306 (Pl.), 832 (Y.F.)
Joseph, M. G., 4 (Lep.)
Joukoff, N. M. & Krassikova, V., 178 (Mal.)
Joukow-Werejnikow, N. & Lipatova, T., 886 (Pl.)

Journal of the Royal Naval Medical Service, 84 (Y.F.), 660 (K.A.)
Journe, H., 812 (Hel.)
Joyeux, C., 387 (Hel.)
——, Baer, J. & Martin, R., 387 (Hel.)
——, with Bonnal & Bosch, 120 (Hel.)
——, Houdemer, E. & Baer, J., 406 (Oph.), 782 (Hel.)
Jude, L. V. R. & Lubet, J. V. H., 880 (Pl.)
Julianelle, L. A. & Harrison, R. W., 401 (Oph.)
Junès, E., 15 (Oph.)
da Justa, A. A., (13) (Lep.)

K

Kadaner, with Schwetz, 817 (Misc.)
Kadaner, M. & Corti, E., 92 (Lept.)
Kalantarian, H., 395 (Hel.)
Kampmeier, R. H. & Hinman, E. H., (824) (Misc.)
Kan, H. C., with Khaw, 681 (Mal.)
Kanagarayer, K., (319) (Der.)
Kanda, K. & So, K., 18 (Oph.)
Karalis, with Petzetakis, 250 (Fev.)
Karpichina, A. & Stein, 538 (Lep.)
Kassirsky, I. A., 658 (K.A.)
Kassirsky, J. A., 86 (R.F.), 231 (K.A.)
——, Burowa, C. F. & Umidowa, S. J., 474 (Mal.)
Kauntze, W. H. & Symes, C. B., 155 (Mal.)
Kawamura, R., Imagawa, Y. & Itô, T., 251 (Fev.)
Keil, E. C., 544 (Lep.)
Kellaway, C. H., 100 *bis* (Sn.)
——, Cherry, R. O. & Williams, F. E., 100 (Sn.)
—— & Holden, H. F., 99 (Sn.)
—— & Williams, F. E., 101, 103 (Sn.)
Keller, A. E., Leathers, W. S. & Ricks, H. C., 795 (Hel.)
Kellogg, W. H., 878 (Pl.)
Kemp, H. A., Moursund, W. H. & Wright, H. E., 86, 508 (R.F.)
Kerbler, F., 147 (Rab.)
Kerr, J. A., 502 (Y.F.)
—— & Patiño Camargo, L., 495 (Y.F.)
Khalil, M., 112, 764 (Hel.), 327 (Misc.)
Khan, S., 890 (Chl.)
——, with Asheshov, I. N., Asheshov, I. & Lahiri, 40 (Chl.)
——, with ——, ——, —— & Chatterji, 41 (Chl.)
Khartoum, Kitchener School of Medicine, 75 (B.R.)
Khaw, O. K. & Kan, H. C., 681 (Mal.)
Kieser, J. A., 771 (Hel.)
Kikuth, W. & Giovannola, A., 175 (Mal.)
Kilborn, L. G., with Cunningham, Maxwell, Morse, Mullett & Dickinson, 323 (Misc.)
Kilduffe, R. A., 395 (Hel.)
Kimura, R., Misugi, Y., Hirai, T. & Miyazawa, Y., 251 (Fev.)
King, H. H. & Iyer, P. V. S., 30 (Pl.)
King, R. L., with Mulholland, 25 (Pel.)
Kingo, S., (855) (Lept.)
Kingsbury, A. N., 153 (Mal.), 728 (Misc.)
Kirby, D. B. & Wiener, R. v. E-, 860 (Oph.)
Kiribayashi, S., (47) (Chl.), 118 (Hel.)
—— & Aida, T., (314) (Chl.)
Kirwan, E. O'G., 17 (Oph.)
Kissmeyer, A., 258 (Lep.)
Klarenbeek, A. & Schüffner, W. A. P., (96) (Lept.)
Kleeberg, J., 239 (Fev.)
Kleine, F. K., (47), 311 (Chl.)
Klerks, J. V., with Radsma & Streef, 362 *bis*, 669 (Misc.)
Kligler, I. J., (436) (Mal.)
—— & Aschner, M., 624 (Fev.)
—— & Mer, G., 423 (Mal.)
Knap, C. R., (13) (Lep.)
Kobayashi, Y., 747 (Misc.)
Koch, F. & Vohwinkel, K. H., 179 (Mal.)
Kodama, M., with Takahashi, 242 (Fev.)
Koechlin, D , with Chorine, 705 (Mal.)
Koldajew, B., with Pikul, 638 (Rab.)
Kolka, P. V. G., 477 (Bb.)
Kolle, W. & Prigge, R., 331 (Misc.)
Konné, M. & Pieraerts, G., 584 (S.S.)
Koo, C., with Wakeshima, 280 (Am.)
Kopciowska, L., with Nicolau, 152, 637, 641 (Rab.)
——, with ——, Balmus & Mathis, 843 (Y.F.)
——, with —— & Mathis, 499, 842, 843, 844, (Y.F.)
——, with ——, —— & Baffé, O., 842 (Y.F.)
Koppisch, E., with Carrión, 316 (Der.)
Kopstein, F., 99 (Sn.), 360 (Misc.)
Koreyeda, T., with Shoda & Otomo, 899 (Chl.)
Korke, V. T., 134, 398 (Hel.)
Kornel, G., 11 (Lep.)
Korteweg, P. C., 165 (Mal.)
Kotrba, J., with Fischl & Singer, 510 (R.F.)
——, with Singer & Fischl, 851 (R.F.)
Kraemer, M., with Asher, 289 (Am.)
Kramer, P. H., (96) (Lept.)
Krassikova, V., with Joukoff, 178 (Mal.)
Krause, M., with Kunert, 597 (S.S.)
Krijgsman, B. J., 214 (S.S.), 354 (Misc.)
Krikorian, K. S., with Getzowa & Stuart, 150 (Rab.)
Krishnan, K. V., (189) (Mal.), 233 (K.A.)
——, Lal, C. & Napier, L. E., 181 (Mal.), 233 (K.A.)
——, with Napier, 225 (K.A.)
——, with —— & Lal, 233 (K.A.)
——, with —— & Smith, 228, 229, 663 (K.A.)
——, Smith, R. O. A. & Lal, C., 179, 463 (Mal.)
Krishnaswamy, R., with David & Rajamanikam, 821 (Misc.)
Kritschewski, I. L. & Demidowa, L. W., 181 (Mal.)
—— & Pines, A. I., 698 (Mal.)
—— & Rubinstein, P. L., 241 (Fev.)
—— & Solowiow, N. N., 627 (Fev.)
—— & Sternberg, E. J., (436) (Mal.)
Kröber, F., 347 (Misc.)
Krouch, M., 434 (Mal.)
Krull, W. H., 111 (Hel.)
Kubo, H. & Ku-Lien-Yuan, 312 (Chl.)
Kuipers, I. J. & Stoker, W. J., 414 (Mal.)
Ku-Lien-Yuan, with Kubo, 312 (Chl.)

Kumm, H. W., 502 (Y.F.)
Kunert, H. & Krause, M., 597 (S.S.)
—— & Kunzmann, T., 589 (S.S.)
——, with Schilling, Schreck & Neumann, 213, 586 (S.S.)
Kunzmann, T., with Kunert, 589 (S.S.)
Kupffer, A., 7 (Lep.)
Kuroya, M. & Ono, H., 899 (Chl.)
Kurpanova, M., with Machwiladze, 177 (Mal.)
Kuteischikow, A., Dosser, E. M. & Bernhoff, F. G., 238 (Fev.)
Kutschinsky, W., 788 (Hel.)
Kutumbiah, P., with Iswariah, 479 (Bb.)
Kyu, K., 101 (Sn.)

L

Laake, E. W., Parman, D. C., Bishopp, F. C. & Roark, R. C., 61 (Ent.)
Laas, E., with Mayer & Sonnenschein, 234 (K.A.)
Labernadie, V. & André, Z., 262 (Lep.)
——, with ——, 547 (Lep.)
La Face, L., (436) (Mal.)
Lafitte, A., with Harvier & de Brun, (726) (Mal.)
Lahiri, B. N., with Shortt, 641 (Rab.)
Lahiri, M. N., with Asheshov, I. N., Asheshov, I. & Chatterji, 42 (Chl.)
——, with ——, —— & Khan, 40 (Chl.)
——, with ——, ——, —— & Chatterji, 41 (Chl.)
Lahiri, S. C., 343 (Misc.)
Lai, D. G., 261 (Lep.)
Laigret, J., 79, 84, 502, 838 (Y.F.), 554 *bis* (Lep.)
—— & Durand, R., 246, 623 (Fev.)
——, with Nicolle, 246, 247, 626 (Fev.)
——, with —— & Giroud, 244 (Fev.)
——, with —— & Sparrow, 630 (Fev.)
——, with Sellards, 501 (Y.F.)
Lajos, S., 474 (Mal.)
Lal, C., with Krishnan & Napier, 181 (Mal.), 233 (K.A.)
——, with —— & Smith, 179, 463 (Mal.)
——, with Napier & Krishnan, 233 (K.A.)
Lal, R. B. & Shah, K. S., 410 (Mal.)
Lal, S. & Dogra, J. B., 235 (K. A.)
Lalazarow, G., with Novikova, 883 (Pl.)
Lambert, S. M., 733 (Misc.)
Lamborn, W. A., 602 (S.S.)
Lampe, P. H. J., 5, 543, 544, 864 (Lep.)
Landeiro, F., 427, (436), 458 (Mal.), 873 (Lep.)
Lane, C., 489 (Pel.)
Lane, J., with Antunes, (823) (Misc.)
de Langen, C. D., (189) (Mal.), 329 (Misc.)
—— & Eerkens, J. W., 125 (Hel.)
Langeron, M., 364 (B.R.)
Lapage, G., 389 (Hel.)
Larsen, A. E., (824) (Misc.)
Lasnet, (189) (Mal.)
Layani, E., with Lemierre & Pham-Huu-Chi, 620 (Fev.)
League of Nations, Health Organisation, 421 (Mal.)
Leake, C. D., with Anderson & Emerson, 868 (Lep.)
——, with David, Johnstone & Reed, 283 (Am.)
Leake, C. D., with Emerson & Anderson, 262 *bis*, 263 (Lep.)
Leathers, W. S., with Keller & Ricks, 795 (Hel.)
Lebedewa, E., with Lugowaja, (888) (Pl.)
Lebon, 648 (Rab.)
Le Chuiton, F. & Moureau, M., 97 (R.B.F.)
Lee, C. U. & Chung, H., 780 (Hel.)
Lefèvre, (361) *bis* (Misc.)
Lefrou, G., 115 (Hel.), 359 (Misc.)
—— & Bonnet, P., 552 *ter*, 867 *bis*, 873 (Lep.)
——, with Des Essarts, 550, 867 (Lep.)
Lega, G. & Casini, G., (189) (Mal.)
Le Gac, P., with Raynal, 60 (Ent.), 231 (K.A.)
Legendre, F., (436), 713 (Mal.)
Legendre, F. M. A. & Razafinjato, 779 (Hel.)
Legendre, J., 742 (Misc.)
Leger, M., 32 (Pl.), 543 (Lep.), 795 (Hel.)
Legroux, C., with Catanei, 316 (Der.)
Leiper, R. T., 372 (Hel.)
Leiserman, L., 698 (Mal.)
Le Mer, with Lemoine, (636) (Fev.)
Lemierre, A., 636, (636) (Fev.)
——, Layani, E. & Pham-Huu-Chi, 620 (Fev.)
Lemoine & Le Mer, (636) (Fev.)
Lemoine, J., with Marchal, 809 (Hel.)
Le Moult, with Pirot, 356 (Misc.)
Lena, D., with Pieri, 235 (K.A.)
Lentjes, L. J. M., 603 (Bb.)
——, with Elshout, 477 (Bb.)
Léon, R., (361) (Misc.)
de Leon, W., Garcia, A. & de Jesus, P. I., 350 (Misc.)
——, with de Jesus, 350 (Misc.)
——, with —— & Anzures, 350 (Misc.)
——, with —— & Ramos, 350 (Misc.)
Lépine, P., 239 (Fev.)
—— & Bilfinger, F., 309 (Pl.), 618, 623 (Fev.)
——, Markianos, J. & Bilfinger, F., 871 (Lep.)
Leprosy in India, 2, 256, 540, 863 (Lep.)
Leprosy Review, 2, 258, 259, 540, 863 (Lep.)
Lereboullet, J., with Guillain, Bertrand & Mollaret, 903 (Pel.)
Le Roy des Barres, A., 390 (Hel.)
Lester, H. M. O., 198, 569 (S.S.)
——, with Lloyd, Taylor & Thornewill, 66 (Ent.)
Lestoquard, F., with Parrot & Donatien, 229 (K.A.)
—, with Sergent, Edm., Sergent, Et., Parrot & Donatien, 226 (K.A.)
Levaditi, C. & Schoen, R., 145 (Rab.)
——, —— & Levaditi, J., 638 (Rab.)
——, —— & Vaisman, A., 855 (R.B.F.)
Le-van-Phung, with Montel, Truong-van-Que & Nguyen-Ngoc-Nhuan, 869 (Lep.)
——, with ——, Tran-van-Hanh, Nguyen-van-Khai, Truong-van-Que & Nguyen-Ngoc-Nhuan, 869 (Lep.)
Lever, R. J. A. W., (72) (Ent.)
Levie, L. H., with de Graaf, 261 (Lep.)
Lewis, D. J., 54 (Ent.)
Lewis, E. A., 62 (Ent.)
Lewis's Medical and Scientific Library, 604 (B.R.)
Li, H. C., 788, 803 (Hel.)
Liang, W., with Bernard & Raynal, 43 (Chl.)

Liang-Yu, W., with Riley, 55 (Ent.)
Lichtwardt, H. A., 864 (Lep.)
Lièvre, H., 376, 769, 791 (Hel.), (436) (Mal.)
——, with Senevet, 769 (Hel.)
Lima, E. de Q., 637 (Rab.)
Lin, L. C., with Struthers, Chang & Ch'en, 662 (K.A.)
Lingen, F. F. L., 93 (Lept.)
Linton, R. W., Mitra, B. N. & Shrivastava, D. L., 892, 893 (Chl.)
—— & Shrivastava, D. L., 39, 313 *bis* (Chl.)
——, —— & Mitra, B. N. 313 (Chl.)
Liotier, R., 793 (Hel.)
Lipatova, T., with Joukow-Werejnikow, 886 (Pl.)
Lloyd, C. H., 105 (Sn.)
Lloyd, L., Lester, H. M. O., Taylor, A. W. & Thornewill, A. S., 66 (Ent.)
Lloyd, W., with Davis & Frobisher, Jr., 81 (Y.F.)
—— & Mahaffy, A. F., 498, 838 (Y.F.)
——, with —— & Penna, 78 (Y.F.)
——, Penna, H. A. & Mahaffy, A. F., 81 (Y.F.)
Lobel, L. W. M., 907 (B.R.)
Lodato, G., 156 (Mal.)
Loëwenstein, E., 8 (Lep.)
Loewenthal, L. J. A., 317 (Der.)
——, with Gibbins, 399 (Hel.)
Löffler, E. & Schweinburg, F., 640 (Rab.)
van Loghem, J. J., 305 (Pl.)
Löhe, H., 666 (K.A.)
Lorando, N., 857 (R.B.F.)
Loré, A. & Marty, J., 202 (S.S.)
Lörincz, F., 120 (Hel.)
—— & Szentkirályi, Z., 235 (K.A.)
Lovaglio, R., (436) (Mal.)
Low, G. C., (361) (Misc.)
Lowe, G. H., 154 (Mal.)
Lowe, J., 865, 872 (Lep.)
——, 3, 260 (Lep.), 26 (Pel.), 420 (Mal.)
Lubet, J. V. H., with Jude, 880 (Pl.)
Luc, R. H. L., with Barral, 14 (Oph.)
Lucherini, T., (189) (Mal.)
Lugowaja, L. & Lebedewa, E., (888) (Pl.)
Lulofs, P. K. D., (361) (Misc.)
Lumbroso, U., 401 (Oph.)
——, with Nicolle, 401 (Oph).
—— & Thygeson, P., 401 (Oph.)
Lund, C. C. & Ingham, T. R., 281 (Am.)
Lupu, E., with Slatineanu, Nitulescu, Sibi & Cantacuzino, 904 (Pel.)
Lush, D., with Farran-Ridge, 285 (Dys.)
Lutrario, A., 167, 416, 686 (Mal.)
Lutz, A., 779 (Hel.)
Lwoff, M., 353 (Misc.)
Lyle, T. K. & Fenton, F. G., 862 (Oph.)
Lynch, H. J., with Christensen, 375 (Hel.)

M

Mabalay, E., with Rodriguez & Tolentino, 265, (874) (Lep.)
McArthur, A. W., with Vinks, 397 (Hel.)
MacArthur, W. P., 119, 384 (Hel.)
McBroom, J., with Jordan, 669 (Misc.)
MacCallan, 402, 403 (Oph.)
McCoy, G. W., (655) (Am.)
McCoy, O. R., 802 (Hel.)
Macdouall, J. C. S., 168 (Mal.)
McGuire, C., 339 (Misc.)
——, with Shortt, Malone & Craighead, 642 (Rab.)
MacHattie, C., Mills, E. A. & Chadwick, C. R., 114 (Hel.)
Macht, D. I. & Davis, M. E., 102 (Sn.)
Machwiladze, N. & Kurpanova, M., 177 (Mal.)
Mackay, R., 427 (Mal.)
——, Buchanan, J. C. R. & Sanderson, I., 470 (Mal.)
Mackehenie, D., with Monge, 254 (Fev.)
McKendrick, A. G., 644 (Rab.)
Mackerras, M. J., 65 (Ent.)
Mackie, F. P., 814 (Misc.)
Mackie, T. T. & Nauss, R. W., 649 (Am.)
McKinley, E. B. & Verder, E., 265 (Lep.)
McKittrick, L. S., (290) (Am.)
Maclean, G., 219 *bis* (S.S.)
MacLeod, J., 747 (Misc.)
MacMahon, E. J. R., 427 (Mal.)
McMullen, 37 (Chl.), 304 (Pl.)
McNair, J. S., with Cooke, (726) (Mal.)
Macpherson, A., 561 (S.S.)
McQueen, W. B., (726) (Mal.)
Magalhães, O., with Moreira, (252) (Fev.)
Magath, T. B., 116 (Hel.)
Mahaffy, A. F., with Beeuwkes, 829 (Y.F.)
——, with Lloyd, 498, 838 (Y.F.)
——, —— & Penna, H. A., 78 (Y.F.)
——, with —— & ——, 81 (Y.F.)
Mahler, K., 107 (Hel.)
Maitland-Jones, A. G., 814 (Misc.)
Maitra, G. C., Naidu, B. P. B. & Ahuja, M. L., 104 (Sn.)
Majid, S. A. & Sinton, J. A., 182 (Mal.)
Majumdar, N. R., with Gupta & Das, 411 (Mal.)
Makehennie, D., 253, 254 (Fev.)
Malacca Agricultural Medical Board, 678 (Mal.)
Malloch, J. R., 61 (Ent.)
Malone, R. H., with Shortt, Craighead & McGuire, 642 (Rab.)
Manai, A., 235 (K.A.)
Manako, K., (314) (Chl.)
Manalang, C., 866 (Lep.)
Manca, S., (189) (Mal.)
Manceaux, A., with Sergent & Balliste, 87 (R.F.)
Mandillon, G. A. L., with Courboulès, (823) (Misc.)
Manson-Bahr, P., (190) (Mal.), 814 (Misc.)
—— & Walters, A. H., 429 (Mal.)
Manteufel, P. & Dressler, I., 92 (R.F.)
Manwell, R. D., 428 *bis* (Mal.)
Maplestone, P. A., 119, 763 (Hel.)
—— & Chopra, R. N., 374, 375 (Hel.)
—— & Mukerji, A. K., 393 (Hel.)
Marcandier, with Plazy & Pirot, (636) (Fev.)
Marchal, G. & Lemoine, J., 809 (Hel.)
Marchoux, E., 866 (Lep.)
—— & Chorine, V., 89 (R.F.)
de Maria, G., with Savignoni, (400) (Hel.)
Marie, A. C., 641 (Rab.)
Marino, P., with Missiroli, 473 (Mal.)
Marino-Assereto, P., with Paladino-Blandini, 713 (Mal.)

Markianos, J., 6 (Lep.)
——, with Lépine & Bilfinger, 871 (Lep.)
Marque, 324 (Misc.)
de Marqueissac, H., 208, 571 (S.S.)
Marras, F. M. & Omar, W., 895 (Chl.)
Marsh, E. B., 786 (Hel.)
Marsh, F., 193 (H.S.)
Martin, with Monnier, 708 (Mal.)
Martin, P., with Morin, 469 (Mal.)
——, with —— & Bader, 470 (Mal.)
Martin, R., Chassigneux & Rouëssé, 689 (Mal.)
——, with Joyeux & Baer, 387 (Hel.)
Martini, E., (73) (Ent.), 416, (726) (Mal.)
——, Mayer, F. & Weyer, F., 59 (Ent.)
Marty, J., with Loré, 202 (S.S.)
Masia, A., with Pinelli, (190) (Mal.)
Mason, M., 222 (B.R.)
Massias, C., 20, 481 (Bb.), 38 (Chl.), 430, 697 (Mal.), (824) (Misc.)
Maternowska, I., 131 (Hel.)
——, with Trawiński, 802 (Hel.)
Mathis, C. & Berland, L., 71 (Ent.)
—— & Durieux, C., 505, 849 (R.F.)
——, —— & Advier, M., 87, 505 (R.F.)
——, with Nicolau & Constantinesco, 145 (Rab.)
Mathis, M., 497, 498, 839, 845 (Y.F.)
—— & Baffet, O., 845 (Y.F.)
——, with Nicolau & Kopciowska, 499, 842, 843, 844 (Y.F.)
——, with ——, —— & Baffé, 842 (Y.F.)
——, with ——, Kopciowska & Balmus, 843 (Y.F.)
Matilla, V., 147 (Rab.)
Matsumoto, K., 391 (Hel.)
Mattlet, G., 268 (Y. & S.)
Mauritius, 764 (Hel.)
Maxwell, J. L., 1 (Lep.)
——, with Cunningham, Kilborn, Morse, Mullett & Dickinson, 323 (Misc.)
Mayer, F., with Martini & Weyer, 59 (Ent.)
Mayer, M., 170, 696 (Mal.)
——, Laas, E. & Sonnenschein, C., 234 (K.A.)
Mayfield, C. R., with Havens, 147 (Rab.)
Mazumdar, T. N., 20 (Bb.)
Mededeelingen van den Dienst der Volksgezondheid in Nederlandsch-Indië, (362) (Misc.)
Medulla, C., (92), (853) (R.F.), (96) (Lept.)
Meersseman, F., Friess, E. & Bergondi, J., 111 (Hel.)
Meggitt, F. J., 781 (Hel.)
Mehta, D. R., 721 (Mal.)
de Meillon, B., 467, 715 (Mal.)
van Meirhaeghe, A., 242, 616 (Fev.)
Meleney, H. E., 355 (Misc.)
——, with Frye, 284 (Am.)
Mellanby, K., 68 (Ent.), 740 (Misc.)
——, with Buxton, 739 (Misc.)
de Mello, F., 679 (Mal.)
de Mello, I. F., 186 (Mal.), (824) (Misc.)
Mendes, A. B. C. & Ferreira, J. C., 657 (K.A.)
de Mendonça, F. C., 654 (Dys.)
de Mendonça, J. I., (483) (Bb.)
Menk, W., with Schlossberger, 103 (Sn.)
Menon, K. P., with Pandit & Iyer, 33 (Pl.)
Menon, T. B., 752 (Misc.)
—— & Annamalai, D. R., 426 (Mal.)
Mer, G., 455 (Mal.)
——, with Kligler, 423 (Mal.)
Mercier, H., with Moustardier, Sicé & Varneau, 573 (S.S.)
Mertens, W. K., 54 (Ent.)
—— & van Veen, A. G., 816 (Misc.)
Mesnard, J. & Toumanoff, C., 717 (Mal.)
Mesquita, M., with Vieira, 633 (Fev.)
Meyer, K. F., 878 (Pl.)
Mezquita Lopez, M. & Garcia Corselas, T., (400) (Hel.)
Michelsen, J., with Talbott, 191 (H.S.)
Mickaniewski & Hai, 690 (Mal.)
Migluelote-Vianna, M., with Vellard, 101 (Sn.)
Miller, D. K. & Rhoads, C. P., 140 (Sp.)
Miller, H. M., Jr., 386 (Hel.)
Millous, (824) (Misc.)
Mills, C. A. & Ogle, C., 192 (H. S.)
Mills, E. A., with MacHattie & Chadwick, 114 (Hel.)
Milne, J. C., 678 (Mal.)
Miloslavski, M., 698 (Mal.)
Mira, M. G., 811 (Hel.)
Missiroli, A., 157 (Mal.)
——, with Christophers, 158 (Mal.)
—— & Marino, P., 473 (Mal.)
—— & Mosna, E., 693 (Mal.).
Misugi, Y., with Kimura, Hirai & Miyazawa, 251 (Fev.)
Mitamura, T., with Nagayo, Hazato & Tamiya, 242 (Fev.)
Mitra, B. N., with Linton & Shrivastava, 313, 892, 893 (Chl.)
Mittelmann, G., 782 (Hel.)
Miyahara, H., with Morishita & Ishioka, (190) *ter*, 694 (Mal.)
Miyazawa, M., with Nishibe & Hosono, 251 (Fev.)
Miyazawa, Y., with Kimura, Misugi & Hirai, 251 (Fev.)
Miyoshi, S., 11 (Lep.)
Mladenow, I., with Tschilow, 703 (Mal.)
Mochtar, A., 93 (Lept.)
Molesworth, E. H., 545 (Lep.)
Molina, R. R., Hoffman, W. A. & Rojas, E. S., (813) (Hel.)
Mollaret, with Giraud & Monges, 660 (K.A.)
Mollaret, P., with Guillain, Bertrand & Lereboullet, 903 (Pel.)
Momma, K., 792 (Hel.)
Monacelli, M., 664, 665 (K.A.)
Monauni, J., 488 (Pel.)
Moncorps, C. & Bohnstedt, R. M., 581 (S.S.)
Mondolfo, U., with Giovanardi, 765 (Hel.)
Monge, C. & Mackehenie, D., 254 (Fev.)
Monges, F., with Giraud & Mollaret, 660 (K.A.)
Monier, H. (436) *ter* (Mal.)
Monnerot-Dumaine, M., (726) (Mal.)
Monnier & Martin, 708 (Mal.)
Monnier, E., with Morin, Bader & Moreau, 459 (Mal.)
Mönnig, H. O., 767, 771 (Hel.), 906 (B.R.)
Monserrat, C., 273 (Y. & S.)
Montañes, P., (874) (Lep.)
—— & Negro (E.), 545, (555) (Lep.), 665 (K.A.)
Montastruc, E., with Guilliny, 13 (Lep.)

de Monte, A. J., with Pasricha & Gupta, 42 (Chl.)
Monteiro, J. L., 250 (bis), 633, 636 (Fev.), (504) (Y.F.)
——, with Do Amaral, 616 (Fev.)
—— & Travassos, J., (504) (Y.F.)
Montel, M. L. R., 869, 873 *bis* (Lep.)
——, Le-van-Phung, Truong-van-Que & Nguyen-Ngoc-Nhuan, 869 (Lep.)
——, Tran-van-Hanh, Nguyen-van-Khai, Le-van-Phung, Truong-van-Que & Nguyen, Ngoc-Nhuan, 869 (Lep.)
Montel, R., 548 (Lep.)
Montestruc, E., with Chorine & Guilliny, (873) (Lep.)
Montoliu Volant, C., with Sanchis Bayarri, 658 (K.A.)
Montus, with Giraud & Ciaudo, 231 (K.A.)
Moorthy, V. N., with Stewart, 49 (Ent.)
Mooser, H. & Sparrow, H., 246 (Fev.)
——, Varela, G. & Pilz, H., 621 (Fev.)
Mora, D., 576 (S.S.)
Morales Gonzalez, J. L., 656 (K.A.)
Morax, V., 402 (Oph.)
Moreau, with Toullec, (475) (Mal.)
Moreau, P., 340 (Misc.)
——, with Morin, Bader & Monnier, 459 (Mal.)
——, with —— & Toumanoff, (190) (Mal.)
Moreira, J. A. & Magalhães, O., (252) (Fev.)
Moretti, P., (190) (Mal.)
Morgan, M. T., 889 (Chl.)
Morgan, W. G., with Walsh, 71 (Ent.)
Moriguchi, T., 515 (Lept.)
Morin, H. G. S., 680 (Mal.)
—— & Bader, H., 154, 466, 718 (Mal.)
——, —— & Martin, P., 470 (Mal.)
——, ——, Monnier, E. & Moreau, P., 459, (Mal.)
——, with Martin, P., 469 (Mal.)
——, Moreau, P. & Toumanoff, C., (190) (Mal.)
Morishita, K., Miyahara, H. & Ishioka, H., (190) *ter*, 694 (Mal.)
Morison, 37 (Chl.)
Morison, J., 314 (Chl.)
——, Rice, E. M. & Pal Choudhury, B. K., 891 (Chl.)
Moroder, J., (603) (S.S.)
Morrison, W. K., 385 (Hel.)
Morrow, T. L. & Wieand, W. G., 168 (Mal.)
Morse, W. R., with Cunningham, Kilborn, Maxwell, Mullett & Dickinson, 323 (Misc.)
Morson, C., 142 (B.R.)
Mortoatmadjo, B., 867 (Lep.)
Morton, T. C. St. C., 191 (H.S.)
Moser, J. R., with Salle, 553 *bis* (Lep.)
Moshkovski, S. D. & Nossina, W., 745 (Misc.)
Mosing, H., with Chrzanowski, 249 (Fev.)
Moskalewa, T. A., with Isabolinski, Sobolewa Stratanowitsch & Riwkina, 619 (Fev.)
Mosna, E., with Missiroli, 693 (Mal.)
Mosteller, R., with Pund, 802 (Hel.)
Motais, 14 (Oph.)
Motais, F., 859 (Oph.)
Mouchet, R., van Hoof, L., Duren, A., Fontana, L., Clarebout, G., Henry, E. & Henrard, C., 831 (Y.F.)
Moufel, P. & Andreeva-Budylina, V., 704 (Mal.)
Mountjoy, J. E., 503 (Y.F.)
Moureau, M., with Le Chuiton, 97 (R.B.F.)
Moursund, W. H., with Kemp & Wright, 86, 508 (R.F.)
Moustardier, G., Sicé, A., Mercier, H. & Varneau, 573 (S.S.)
Moyne, with Riou, 202 (S.S.)
Mrugowsky, J., 84 (Y.F.)
Mudaliyar, M. R. G., Sundaram, S. K. & Ramachandran, A. S., 230 (K.A.)
Mühlens, P., 232 (K.A.), 514 (R.F.), 694 (Mal.)
Muir, E., 546 (Lep.)
—— & Chatterji, S. N., 549 (Lep.)
—— & Chatterji, S. P., 861 (Oph.)
Mukerji, A. K., with Chopra & Ghosh, 768 (Hel.)
——, with Maplestone, 393 (Hel.)
Mukherjee, B., with Chopra & Campbell, 173 (Mal.)
Mukherjee, S. N., with Chopra & Sundar Rao, 808 (Hel.)
Mulholland, H. B. & King, R. L., 25 (Pel.)
Muller, A. R., 859 (Oph.)
Müller, H., with Huisman, 340 (Misc.)
Mullett, H. J., with Cunningham, Kilborn, Maxwell, Morse & Dickinson, 323 (Misc.)
Mulligan, H. W. & Sinton, J. A., 462 (Mal.)
——, with ——, 461 (Mal.)
Murao, K., with Aoki, 266 (Lep.)
Murashima, T., 70 (Ent.)
Musger, A., (813) (Hel.)
Musgrave, A., with Ingram, 71 (Ent.)

N

Nabokoff, V., Gorjainoff, A. & Bekman, A., 57 (Ent.)
Nagayo, M., Hazato, H., Mitamura, T. & Tamiya, T., 242 (Fev.)
Nagayoshi, K., 771 (Hel.)
Nägelsbach, E., 734 (Misc.)
Naidu, B. P. B., 884 (Pl.)
——, with Maitra & Ahuja, 104 (Sn.)
Nair, V. G., with Iswariah, 551 (Lep.)
Nandi, P., with Roy & Ghosh, 20 (Bb.)
Napier, L. E. & Gupta, C. R. D., 661 (K.A.)
—— & Krishnan, K. V., 225 (K.A.)
——, —— & Lal, C., 233 (K.A.)
——, with —— & ——, 181 (Mal.), 233 (K.A.)
—— & Sharma, L. R., 232, 664 (K.A.)
—— & Smith, R. O. A., 661 (K. A.)
——, —— & Gupta, C. R. D., 227 (K.A.)
——, —— & Krishnan, K. V., 228, 229, 663 (K.A.)
Narihara, N., with Wakeshima & Yoshino, 394 (Hel.)
Naritomi, C., 252 (Fev.)
Nash, T. A. M., 65 (Ent.), 216 (S.S.)
Nataf, R., with Cuénod, 861 (Oph.)
Natali, C., 691 (Mal.), 867 (Lep.)
Nattan-Larrier, L. & Girard, G., 276 (Y. & S.)
—— & Grimard-Richard, L., 230, 657 (K.A.)
Nauss, R. W., with Mackie, 649 (Am.)
Navas Torres, A., (436) (Mal.)

Nederlandsche Vereeniging voor Tropische Geneeskunde, 738 (Misc.)
Negro, E., with Montañes, 545, (555) (Lep.), 665 (K.A.)
Neitz, W. O. & Thomas, A. D., 149 (Rab.)
Nelson, E. C., 288 (Dys.)
Neumann, H., (190) (Mal.)
——, with Schilling, 167 (Mal.)
——, with ——, Schreck & Kunert, 213, 586 (S.S.)
Neumann, M. W., with Grunfeld & Serebrjannaja, 622 (Fev.)
——, with Serebrennaja & Grünfeld, 243 (Fev.)
Neveu, R., with Tanon, 72 (Ent.)
Neveu-Lemaire, M., 49 (Ent.)
Nezic, E., 170 (Mal.)
Nezitch, E., 685 (Mal.)
Ngai, S. K. & Frazier, C. N., 651 (Am.)
Nguyen-duy-Ha, with Chesneau, 64 (Ent.)
Nguyen-ngoc-Nhuan, with Montel, Le-van-Phung & Truong-van-Que, 869 (Lep.)
——, with ——, Tran-van-Hanh, Nguyen-van-Khai, Le-van-Phung & Truong-van-Que, 869 (Lep.)
Nguyen-van-Khai, with Montel, Tran-van-Hanh, Le-van-Phung, Truong-van-Que & Nguyen-ngoc-Nhuan, 869 (Lep.)
Nguyen-van-Tung, 272 (Y. & S.)
Nicholas, M. J., 149 (Rab.)
Nicholls, L., 345 (Misc.)
Nicolau, S., with Haguenau & Cruveilhier, 648 (Rab.)
—— & Kopciowska, L., 152, 637, 641 (Rab.)
——, ——, Balmus, G. & Mathis, M., 843 (Y.F.)
——, —— & Mathis, M., 499, 842, 843, 844 (Y.F.)
——, ——, —— & Baffé, O., 842 (Y.F.)
——, Mathis, C. & Constantinesco, V., 145 (Rab.)
Nicolle, C., 244, 625, 628 (Fev.)
—— & Giroud, P., 240 (Fev.)
——, —— & Sparrow, H., 628, 629 (Fev.)
—— & Laigret, J., 246, 247, 626 (Fev.)
——, —— & Giroud, P., 244 (Fev.)
——, —— & Sparrow, H., 630 (Fev.)
—— & Lumbroso, U., 401 (Oph.)
—— & Sparrow, H., 627 (Fev.)
Nieschulz, O., 48, 61 (Ent.)
Nijkamp, J. A. & Swellengrebel, N. U., 710 (Mal.)
Nikolskaja, Z., with Trapesontzewa, 403 (Oph.)
Niño, F. L., (319) (Der.), 789, 801 (Hel.)
Nishibe, M., Hosono, S. & Miyazawa, M., 251 (Fev.)
van Nitsen, R., 267 (Y. & S.)
Nitulescu, J., with Slatineanu, Francke, Sibi & Paraschivescu, 904 (Pel.)
——, with ——, Sibi, Cantacuzino & Lupu, 904 (Pel.)
——, with ——, ——, Francke & Veit, 904 (Pel.)
Nobl, G., 275 (Y. & S.), (874) (Lep.)
Nodenot, L. & Berny, P., 265 (Lep.)
Nolasco, J. O., 547 (Lep.)
Nono, A. M., with Russell, 681 (Mal.)
Noronha, A. J., 350 (Misc.)
Nossina, W., with Moshkovski, 745 (Misc.)
Noury, M., with Blanc & Baltazard, 243, 626 (Fev.)
——, with ——, —— & Barnéoud, 248 (Fev.)
——, with ——, —— & Fischer, 87 (R.F.)
Novikova, E., & Lalazarow, G., 883 (Pl.)

O

O'Connor, F. W., with Golden, 399 (Hel.)
—— & Hulse, C. R., 133 (Hel.)
d'Oelsnitz, 657, 661 (K.A.)
—— & Carcopino, E., 232 *bis* (K.A.)
—— & Ronchése, A. D., 658 (K.A.)
Oesterlin, M., with Giemsa, 174 (Mal.)
Office International d'Hygiéne Publique, Paris, 889 (Chl.)
O'Flynn, J. A., with Gibson, 660 (K.A.)
—— & Rudd, E. T. S., (824) (Misc.)
Ogle, C., with Mills, 192 (H.S.)
Okuwada, M., 148 (Rab.)
Olmer, D., 387 (Hel.)
Omar, W., 896 (Chl.)
——, with Marras, 895 (Chl.)
Ono, H., with Kuroya, 899 (Chl.)
Orenstein, A. J., 793 (Hel.)
Ori, A., 33 (Pl.)
Oropeza, P., 648 (Rab.)
Ortega Nieto, L., 257 (Lep.)
Oshiro, T., with Aoki, 892 (Chl.)
Otomo, T., with Shoda & Koreyeda, 899 (Chl.)
Otten, L., 35 *bis* (Pl.)
Otto, G. F., with Cordi, 395 (Hel.)
—— & Cort, W. W., 789 *bis* (Hel.)
Otto, R., 617 (Fev.)
Ottolenghi, D., (726) (Mal.)
——, with Cantacuzéne, Swellengrebel & Pittaluga, 162 (Mal.)
—— & Rosa, A., (726) (Mal.)
Ouchi, T., 37 (Chl.)
Owen, H. B., 16 (Oph.)
Oyarzabal, J. D., with Zuccarini, 217 (S.S.)

P

Paine, R. W., 807 (Hel.)
Pakiam, A. X., 382 (Hel.)
Paladino-Blandini, A., (73) (Ent.)
—— & Marino-Assereto, P., 713 (Mal.)
Palant, B. L., with Tzekhnowitzer, 631 (Fev.)
Pal Choudhury, B. K., with Morison & Rice, 891 (Chl.)
Paldrock, A., 538, (555) (Lep.)
—— & Pooman, A., 549 (Lep.)
Pales, 350 (Misc.)
—— & Pouderoux, (362) (Misc.)
Pales, L., 572 (S.S.)
Pallary, P., (400) (Hel.)
Pallister, R. A., 396 (Hel.)
Palthe, van W., 668 (Misc.)
Pampana, E. J., (725) (Mal.)
Panagia, A., 182 (Mal.), (824) (Misc.)
Panayotatou, A., 245 (Fev.)
Pandit, C. G., Menon K. P. & Iyer, P. V. S., 33 (Pl.)
Parada Gay, M. A., with Varela, 631 (Fev.)

Paraschivescu, Z., with Slatineanu, Nitulescu, Francke & Sibi, 904 (Pel.)
Pardal, E., 304 (Pl.)
Pardina, J. M., (813) (Hel.)
Parker, R. R., with Davis, 249, 633 (Fev.)
——, with —— & Walker, 634 (Fev.)
Parman, D. C., 50 *bis* (Ent.)
——, with Laake, Bishopp & Roark, 61 (Ent.)
Paroni, G., 280, (290) (Am.)
Parrino, G., 312 (Chl.)
Parrot, L., (824) *bis* (Misc.)
——, Donatien, A. & Lestoquard, F., 229 (K.A.)
——, with Sergent, E., Sergent, Et. & Donatien, 423 (Mal.)
——, with Sergent, Et., Sergent, Edm., Donation & Lestoquard, 226 (K.A.)
Parulkar, R. D., 307 (Pl.)
Pasco, A. M., with Tubangui, 378 (Hel.)
Pasqual, J. H., 68 (Ent.)
Pasricha, C. L., de Monte, A. J. & Gupta, S. K., 42 (Chl.)
Patiño Camargo, L., with Kerr, 495 (Y.F.)
Paton, J. T., with Colvin & Dawes, (13) (Lep.)
Patrikios, I. & Catacouzenos, E., 150 (Rab.)
Patto, O. & Carneiro, J. M., 641 (Rab.)
Patton, W. S., 55 (Ent.), 743, 744 (Misc.)
——, with Cushing, 359 (Misc.)
Paulino, P. H., with Albert, 793 (Hel.)
Pavlova, P., 700 (Mal.)
Pavlova, P. I., 468 (Mal.)
Payne, F., with Ashford & Payne, G. C., 122, 391 (Hel.)
Payne, G. C., with —— & Payne, F. K., 122, 391 (Hel.)
Payne, S. A. & Perlzweig, W. A., 491 (Pel.)
Peaston, H., 382 (Hel.)
Pecori, G. & Escalar, G., 186 (Mal.)
Peel, with Schwetz & Baumann, 173 (Mal.)
——, with ——, —— & Belhommet, 410 (Mal.)
——, with ——, —— & Droeshaut, 418, (474) (Mal.)
Pelikan, E. R., with Hart, 887 (Pl.)
Penington, R. G., (362) (Misc.)
Penna, H., with Soper, Cardoso, Serafim, Jr., Frobisher, Jr. & Pinheiro, 77 (Y.F.)
Penna, H. A., with Lloyd & Mahaffy, 81 (Y.F.)
——, with Mahaffy & Lloyd, 78 (Y.F.)
Penso, G., 127, 383 (Hel.), 589 (S.S.), (824) (Misc.)
Perlzweig, W. A., with Payne, 491 (Pel.)
Peschkowsky, J. W., 553 (Lep.)
Peshkov, H., 279 (Am.)
Peter & Gaehtgens, 819 (Misc.)
Peter, F. M. (726) (Mal.)
Petigara, P. M., (726) (Mal.)
Pettit, A. & Stefanopoulo, G. J., 79, 838 (Y.F.)
Petruschewsky, G. K. & Tarassow, V., 121, (Hel.)
Petzetakis & Karalis, 250 (Fev.)
Petzetakis, M., 95, (855) (Lept.)
Peyri, A., (874) (Lep.)
Pezzeri, V., 704 (Mal.)
Pham-Huu-Chi, with Lemierre & Layani, 620 (Fev.)
Phleng, with Tirouvanziam, (727) (Mal.)
Philip, C. B., 503 (Y. F.)
——, with Jellison, 69 (Ent.)
Picard, R., 138 (Sp.)
Piccininni, F., 416 (Mal.)
Pieraerts, G., with Konné, 584 (S.S.)
Pieri, J. & Lena, D., 235 (K. A.)
Pierret, R., 336 (Misc.)
Pijper, A., 771 (Hel.)
—— & Dau, H., 241 (Fev.)
Pikul, I., with Tareev, Epstein, Gontaeva & Raskin, 698 (Mal.)
Pikul, N., with Koldajew, 638 (Rab.)
Pilz, H., with Mooser & Varela, 621 (Fev.)
Pinard, M., (727) (Mal.)
Pinelli, L. & Masia, A., (190) (Mal.)
Pines, A. I., with Kritschewski, 698 (Mal.)
Piney, A., 520 (B.R.)
Pinheiro, J., with Soper, Penna, Cardoso, Serafim, Jr. & Frobisher, Jr., 77 (Y.F.)
Pirot, R. & Le Moult, 356 (Misc.)
——, with Plazy & Marcandier, (636) (Fev.)
Pittaluga, G., with Cantacuzéne, Ottolenghi & Swellengrebel, 162 (Mal.)
Plantilla, F. C., with Rodriguez, 865 (Lep.)
Plantureux, E., 146, 647 (Rab.)
Plantureux, R., 152 (Rab.)
Plazy, Marcandier & Pirot, (636) (Fev.)
Plazy, L. & Damany, P., 120 (Hel.), (252) (Fev.)
Poindexter, H. A., 281 (Am.), 594 (S.S.)
— & Jones, R. F., 804 (Hel.)
Pokrouskaja, M., 882 (Pl.)
Poleff, L., 859 (Oph.)
Poljak, I. & Toumansky, V., 31 (Pl.)
Pollitzer, R., with Wu Lien-Teh, Chun & Wu, 519 (B.R.)
Pons, J. A. & Hoffman, W. A., 381 (Hel.)
——, with —— & Janer, 778 (Hel.)
Pons, R., 308 (Pl.), 817 (Misc.)
—— & Advier, 34 (Pl.)
Pontes, J. de P. L., (727) *bis* (Mal.)
——, with Velho-Da-Silva, 816 (Misc.)
Pookels, W., 823 (Misc.)
Pooman, A., with Paldrock, 549 (Lep.)
Popesco, C., with Combiesco, 242 (Fev.)
——, with ——, Stamatesco & Angelesco, 245 (Fev.)
——, with —— & Vantu, 626 (Fev.)
Popesco-Combiesco, C. & Soru, E., 894 (Chl.)
Popesco, F., 134 (Hel.)
Popoff, with Gontier, 513 (R. F.)
Popow, P. P. & Golzowa, R. D., 49 (Ent.)
Portelly, J., 632 (Fev.)
Porter, A., 750 (Misc.)
Postmus, S., with Snijders & Schüffner, 840 (Y.F.)
Pottier, R., with Van den Branden, 207 (S.S.)
Potts, W. H., 65 (Ent.)
Pouderoux, with Pales (362) (Misc.)
Pousson, A., (362) (Misc.)
Poutchkova, A. I., with Tarejew, 458 (Mal.)
Póvoa, H., (400) (Hel.)
Predteschenskaia, L., with Glusman & Ssolowiewa, 151 (Rab.)
Prendel', A. R., Zagorovskiĭ, N. A. & Futran, G. S., 471 (Mal.)
Previtera, A., 635 (Fev.), 664 (K.A.)
Prigge, R., with Kolle, 331 (Misc.)

Prince, F. M., with Hull & Dove, 745 (Misc.)
Proca, G., Bobes, S. & Jonnesco, D., 642 *bis* (Rab.)
Proceedings of the Royal Society of Medicine, 814 (Misc.)
Prommas, C. & Daengsvang, S., 801 (Hel.)
Prudhomme, R., with Chorine, 867 (Lep.)
Pruis, G. W. A., 122 (Hel.)
Puente, J. J., with Balina, (266) (Lep.)
Pund, E. R. & Mosteller, R., 802 (Hel.)

Q

Quaife, W. T., 711 (Mal.)

R

Rabello, E., (13) (Lep.)
Rabl, R., 334 (Misc.)
Radacovici, E., with Ionesco-Miahaiesti, Zotta & Badenski, 707 (Mal.)
Radhakrishna Rao, M. V., 342 (Misc.)
Radsma, W., Streef, G. M. & Klerks, J. V., (362), 669 *bis* (Misc.)
Raffel, S., 586 (S.S.)
Ragiot & Delbove, (96), 854 (Lept.)
Ragle, B. H., with Urmy, Allen & Jones, 481 (Bb.)
Rajadhyaksha, B. H., (29) (Pel.)
Rajamanikam, N., with David & Krishnaswamy, 821 (Misc.)
Ramachandran, A. S., with Mudaliyar & Sundaram, 230 (K. A.)
Raman, T. K., 26 (Pel.)
Ramirez, J., with Schöbl, Hirano, Vazquez-Colet & Arima, 96 (R. B.F.)
Ramos, J. M., with de Jesus & de Leon, 350 (Misc.)
Ramsay, G. W. St. C., 378 (Hel.)
Rankov, M., 685 (Mal.)
Rao, B. A., (475) (Mal.)
Rao, M. A. N., 378 (Hel.)
Rao, S. S., 134 (Hel.)
Raskin, with Tarejew, Epstein, Gantaeva & Bolotina, 174 (Mal.)
Raskin, A., with Tareev, Epstein, Pikul & Gontaeva, 698 (Mal.)
Ratcliffe, H. L., 287 (Dys.)
Rawlings, I. D., with Bundesen & Fishbein, (289) (Am.)
Ray, N. N., 405 (Oph.)
Raybaud, A., 796 (Hel.), 881 (Pl.)
Raynal, J., with Bernard & Liang, 43 (Chl.)
—— & Le Gac, P., 60 (Ent.), 231 (K.A.)
Razafinjato, with Legendre, 779 (Hel.)
Read, B. E., 548 (Lep.)
Reed, A. C., (655) (Am.)
——, with David, Johnstone & Leake, 283 (Am.)
—— & Johnstone, H. G., 649 (Am,)
Reed, G. B., 10 (Lep.)
Reenstierna, J., 265 (Lep.)
Reeves, R. J. & Apple, E. D., 281 (Am.)
Reichs-Gesundheitsblatt, 149 (Rab.)
Reiner, L. & Chao, S. S., 212 (S.S.)
——, with Smythe, 580 (S.S.)
Reither, R., 716 (Mal.)
Reitler, R., with Scott & Ayoub, 794 (Hel.)
Reitz, H., 549 (Lep.)
Reko, V. A., (13) (Lep.)
Remlinger, P., 145 (Rab.)
—— & Bailly, J., 146 *quat.*, 148, 639, 640, 647 *bis* (Rab.)
Revue de Microbiologie, d'Epidémiologie et de Parasitologie, 882 (Pl.)
Reynal, J. & Wassilieff, A., 35 (Pl.)
Rhoads, C. P., with Miller, 140 (Sp.)
Ribeyro, R. E., 253 (Fev.)
Ricci, E., (824) (Misc.)
Rice, C. E., Drake, A. A. & Smith, J. E., 858 (Oph.)
Rice, E. M., with Morison & Pal Choudhury, 891 (Chl.)
Rickard, E. R., with Soper & Crawford, 836 (Y.F.)
Ricks, H. C., with Keller & Leathers, 795 (Hel.)
Riley, W. A. & Liang-Yu, W., 55 (Ent.)
—— & Scheifley, C. H., 801 (Hel.)
Riou & Moyne, 202 (S.S.)
Riou, M., 340 (Misc.), 400 (Hel.)
——, with Advier, 883 (Pl.)
——, with Advier & Alain, 848 (R.F.)
—— & ——, 235 (K.A.)
——, with Blondin, 340 (Misc.)
Ripley, H. S. & van Sant, H. M., 856 (R.B.F.)
Riseman, J. E. F., with Ernstene, 237 (Fev.)
Rivera Bandres, J., 234 (K.A.)
Rivera, J., 234 (K. A.)
Rivoalen, P., with Sicé & Cousin, 203 (S.S.)
Riwkina, S. L., with Isabolinski, Sobolewa, Stratanowitsch & Moskalewa, 619 (Fev.)
Roark, R. C., with Laake, Parman & Bishopp, 61 (Ent.)
Robatto, A. G., (813) (Hel.)
Robic, J., 32 (Pl.)
——, with Girard, 885 (Pl.)
Robin, L., 154 (Mal.)
Robin, L. A., 412, 414 (Mal.)
—— & Toumanoff, C., 701 (Mal.)
Robineau, M., 539 (Lep.)
Robinson, W. & Simmons, S. W., (824) (Misc.)
Rockefeller Foundation, 222 (B.R.)
Rode, P., 877 (Pl.)
Rodenbeck, 149 (Rab.)
Rodenwaldt, E., 183 (Mal.)
Rodhain, J., 419 (Mal.)
—— & van Hoof, M. T., 377 (Hel.)
Rodieux, M., with Chorine, 457 (Mal.)
Rodrigues, S. L., 8 (Lep.)
Rodriguez, J., (73) (Ent.)
——, Mabalay, E. & Tolentino, J. C., 265, (874) (Lep.)
—— & Plantilla, F. C., 865 (Lep.)
Rogers, L., 349 (Misc.)
Roig I Roig, M., 149 (Rab.)
Rojas, E. S., with Molina & Hoffman, (813) (Hel.)
Romaña, C., 599 (S.S.)
Ronchèse, A. D., with d'Oelsnitz, 658 (K.A.)
Ronka, E. K. F., (813) (Hel.)
Ronse, M., 625 (Fev.)
Roques, H., 14 (Oph.)
Roques, P., with Huard & Dejou, (655) (Am.)
Rosa, A., with Ottolenghi, (726) (Mal.)
Roscoe, M. H., 491 *ter* (Pel.)

Rose, F. G., 539 (Lep.)
Roskott, E. R. A. L., (515) (Lept.)
Rostkowski, L., 402 (Oph.)
Roton, with Souchard, 8 (Lep.)
Roubaud, E., 424, 724 (Mal.)
—— & Colas-Belcour, J., 58 (Ent.), 720 (Mal.)
—— & Treillard, M., 715, 718 (Mal.)
Rouësse, with Martin & Chassigneux, 689 (Mal.)
Rousse, 328 (Misc.)
Row, R., Dalal, N. P. & Gollerkeri, G. V., 172 (Mal.)
Roxburgh, A. C., (13) (Lep.)
Roy, B. C., Nandi, P. & Ghosh, J. L., 20 (Bb.)
Roy, D. N., with Strickland, 178 (Mal.)
de Rozario, C. F., 32 (Pl.)
Rozeboom, L. E. & Shah, K. S., 701 (Mal.)
Ruata, V., 858 (Oph.)
Rubinstein, P. L., with Kritschewski, 241 (Fev.)
Rud, E., 904 (Pel.)
Rudd, E. T. S., with O'Flynn, (824) (Misc.)
Ruffin, J. M. & Smith, D. T., 490 (Pel.)
Rugg-Gunn, A., 405 (Oph.)
Russell, F. F., (504) (Y.F.)
Russell, P. F., 160, (190), 430, (436) (Mal.), 765 (Hel.)
—— & Nono, A. M., 681 (Mal.)
—— & Santiago, D., 720 (Mal.)
Ruys, A. C., 94 (Lept.)
Ryrie, G. A., 8, 542, 864 (Lep.)

S

Sabry, I., 901 (Pel.)
Saenz, A., with Coste & Costil, 276 (Y. & S.)
St. John, J. H., 283 (Am.)
Sainton, J., with Brumpt & Duvoir, 787 (Hel.)
Saliternik, Z., 58 (Ent.)
Salle, A. J., 871 (Lep.)
——, with Emerson, 547 (Lep.)
—— & Moser, J. R., 553 *bis* (Lep.)
Sampedro, M. M., (727) (Mal.)
Sanchez, E., with Vicente Paniagua, (92) (R.F.)
Sanchis Bayarri, V., 289 (Dys.)
—— & Montoliu Volant, C., 658 (K.A.)
Sanderson, I., with Mackay & Buchanan, 470 (Mal.)
Sandosham, A. A., (813) (Hel.)
van Sant, H. M., with Ripley, 856 (R.B.F.)
Santiago, V., (874) (Lep.)
Sanyal, S., 17 (Oph.)
Sapeika, N., 697 (Mal.)
Sardjito, (92) (R.F.)
——, with Bergman & Sitanala, 263 (Lep.)
—— & Sitanala, J. B., 550 (Lep.)
Sarkar, S. L. & Gupta, B. M., 21 (Bb.)
Sarnelli, T., (362) (Misc.)
Sassuchin, D., 359 (Misc.)
Sato, S., 130, (137) (Hel).
Saubidet, E. J., with Cibils Aguirre, (289) (Am.)
Saura Fargas, J., (514) (R.F.)
Sautet, J., 465, 685 (Mal.)
——, with Benelli & Zuccarelli, 660 (K.A.)
——, with Galliard, 722 (Mal.)
——, with Giamarchi, 360 (Misc.)
Santiago, D., with Russell, 720 (Mal.)
Savage, J. de la M., 411 (Mal.)
Savignoni, F. & de Maria, G., (400) (Hel.)
Savino, E., (888) (Pl.)
Sawhney, M. R., 405 (Oph.)
Sawyer, W. A., 837 *bis* (Y.F.)
Sayers, E. G., 821 (Misc.)
Scaduto, P., 393 (Hel.), (636) (Fev.)
Scalas, A., with Tedeschi, 415 (Mal.)
Scamarcio, F. C., with Andreotti, 392 (Hel.)
Scannell, D. D., (290) (Am.)
Scaturro, A., (636) (Fev.)
Schapiro, L., 468 (Mal.)
Scharrer, B., 853 *bis* (R.F.)
Scheifley, C. H., with Riley, 801 (Hel.)
Schennberg, P., with Advier, (219) (S S.)
Schichobalowa, N., with Bransburg & Gorelik, 782 (Hel.)
Schilling, C., 702 (Mal.)
—— & Neumann, H., 167 (Mal.)
——, with Schreck, H., Neumann, H. & Kunert, H., 213, 586 (S.S.)
Schlossberger, H., with Fischl, 518 (B.R.)
—— & Menk, W., 103 (Sn.)
Schlossmann, K., 9 (Lep.)
Schöbl, O., Hirano, H., Vazquez-Colet, A., Ramirez, J. & Arima, S., 96 (R.B F.)
Schoen, R., with Levaditi, 145 (Rab.)
——, with —— & Levaditi, J., 638 (Rab.)
——, with —— & Vaisman, 855 (R.B.F.)
Scholtens, R. T., 312 *bis* (Chl.)
Schottmüller, (141) (Sp.)
Schousboë, 403 (Oph.)
Schreck, H., with Schilling, Neumann & Kunert, 213, 586 (S.S.)
Schüffner, W., 853 (Lept.)
Schüffner, W. A. P., with Klarenbeek, (96) (Lept.)
——, with Snijders & Postmus, 840 (Y.F.)
Schütze, H., 886 (Pl.)
Schwartz, S. C., with Andrews & Johnson, 283 (Am.)
Schweinburg, F., with Löffler, 640 (Rab.)
Schwennicke, H., 783, 788 (Hel.)
Schwetz, J., 91, 509 (R.F.), 461, 462, 681, 708, (727) *bis* (Mal.), 587 (S.S.)
——, Baumann, H. & Peel, 173 (Mal.)
——, ——, —— & Belhommet, 410 (Mal.)
——, ——, —— & Droeshaut, 418, (474) (Mal.)
—— & Kadaner, 817 (Misc.)
Scott, G. W., 712 (Mal.)
Scott, J. A., Ayoub, G. E. & Reitler, R., 794 (Hel.)
Scrimgeour, H., 351 (Misc.)
Scuderi, G., 129 (Hel.)
Sebrell, W. H., 902 (Pel.)
Sedan, J., 16, 402 (Oph.)
Seeley, S. F., 6 (Lep.)
See-Lü, H., 691 (Mal.)
Séguy, E., (362) (Misc.)
Seiffert, W., with Uhlenhuth, 351 (Misc.)
Selfa, E., 859 (Oph.)
Sellards, A. W. & Laigret, J., 501 (Y.F)
Semenova, N. E., 803 (Hel.)

Semikov, F., & Achourova, I., 883 (Pl.).
Sen, B., with Chopra, 652 (Am.)
——, with —— & Sen, S., 652 (Am.)
——, with Sen, S. & Chopra, 282 (Am.)
Sen, S., with Chopra & Sen, B., 282, 652 (Am.)
Senevet, G., 722 (Mal.)
—— & Collignon, E., 156, (727) (Mal.)
—— & Lièvre, H., 769 (Hel.)
Serafim, J., Jr., with Soper, 502 (Y.F.)
——, with ——, Penna, Cardoso, Frobisher, Jr. & Pinheiro, 77 (Y.F.)
Serebrennaja, A. I., Grünfeld, A. A. & Neumann, M. W., 243 (Fev.)
Serebrjannaja, A. I., with Grünfeld & Neumann, 622 (Fev.)
Serefettin, O., 857 (R.B.F.)
Sergent, A., 88 (R.F.)
——, Manceaux, A. & Balliste, R., 87 (R.F.)
——, with Sergent, Et., 361 (Misc.)
—— & Vogt, P., 432, 433, 697 *bis* (Mal.)
Sergent, E., Sergent, Et. & Catanei, A., 418, (436), 704 (Mal.)
——, ——, Parrot, L., Donatien, A. & Lestoquard, F., 226 (K.A.)
——, ——, —— & ——, 423 (Mal.)
Sergent, Et. & Sergent, A., 361 (Misc.)
——, with Sergent, Edm. & Catanei, 418, (436), 704 (Mal.)
——, with ——, Parrot, Donatien & Lestoquard, 226 (K.A.)
——, with ——, —— & ——, 423 (Mal.)
Sfameni, A., 126 (Hel.)
Shah, K. S., with Lal, 410 (Mal.)
——, with Rozeboom, 701 (Mal.)
Shahin, M., 683 (Mal.)
Shapiro, J. M., 683 (Mal.)
Sharma, L. R., with Napier, 232, 664 (K.A.)
Sharp, L. E. S., 542 (Lep.)
Shattuck, G. C., in collaboration with others, 223 (B.R.)
—— & Hilferty, M. M., 192 (H.S.)
Shearer, G. D., 389 (Hel.)
Shiga, K., 286 (Dys.)
Shillong, 314 (Chl.)
Shimojo, K., Soda, T. & Adachi, K., 37 (Chl.)
Shlenova, M. F., (727) (Mal.)
Shoda, T., Koreyeda, T. & Otomo, T., 899 (Chl.)
Shorb, D. A., 117 (Hel.)
Shortt, H. E., 648 (Rab.)
—— & Brooks, A. G., 638 (Rab.)
—— & Lahiri, B. N., 641 (Rab.)
——, Malone, R. H., Craighead, A. C. & McGuire, J. P., 642 (Rab.)
Shrivastava, D. L., with Linton, 39, 313 *bis* (Chl.)
——, with —— & Mitra, 313, 892, 893 (Chl.)
Shute, P. G., 51 (Ent.)
Sibi, M., with Slatineanu, Nitulescu, Cantacuzino & Lupu, 904 (Pel.)
——, with ——, ——, Francke & Paraschivescu, 904 (Pel.)
——, with ——, ——, —— & Veit, 904 (Pel.)
Sicard, M., 513 (R.F.)
Sicault, G., 712 (Mal.)
—— & Decourt, P., 431, 432 (Mal.)
Sicé, A., 31 (Pl.), 577 (S.S.)
Sicé, A., Cousin, E. & Dantec, 577 (S.S.)
——, —— & Rivoalen, P., 203 (S.S.)
——, with Moustardier, Mercier & Varneau, 573 (S.S.)
Silber, L. A. & Dossère, E. M., 623 *bis* (Fev.)
Simić, T., 284 (Am.)
Simmonds, H. W., 358 (Misc.)
Simmons, S. W., with Robinson, (824) (Misc.)
Simons, L. H., 541 (Lep.)
Sinaï, I., 900 (Pel.)
Sincke, G. E., (362) (Misc.)
Singer, E. & Fischl, V., 510 (R.F.)
——, with — —, 851 (R.F.)
——, with —— & Kotrba, 510 (R.F.)
——, Kotrba, J. & Fischl, V., 851 (R.F.)
Singh, B., with Jafar, 255 (Fev.)
Sinton, J. A., 691, 706, (825) (Mal.)
——, with Christophers & Covell, 517 (B.R.)
——, with Majid, 182 (Mal.)
—— & Mulligan, H. W., 461 (Mal.)
——, with Mulligan, 462 (Mal.)
Sitanala, with Bergman & Sardjito, 263 (Lep.)
Sitanala, J. B., with Sardjito, 550 (Lep.)
Slatineanu, A., Nitulescu, J., Francke, M., Sibi, M. & Paraschivescu, Z., 904 (Pel.)
——, ——, Sibi, M., Cantacuzino, L. & Lupu, E., 904 (Pel.)
——, ——, ——, Francke, M. & Veit, E., 904 (Pel.)
Sliwensky, M., 415 (Mal.)
van Slype, W., 250 (Fev.)
Smart, A. G. H., 99 (Sn.), 541 (Lep.)
Smith, D. T., with Ruffin, 490 (Pel.)
Smith, E. C., 497 (Y.F.), (825) (Misc.)
—— & Elmes, B. G. T., 274 (Y. & S.)
Smith, J. E., with Rice & Drake, 858 (Oph.)
Smith, J. H., 490 (Pel.)
Smith, R. O. A., 233 (K.A.)
——, with Krishnan & Lal, 179, 463 (Mal.)
——, with Napier, 661 (K.A.)
——, with —— & Gupta, 227 (K.A.)
——, with —— & Krishnan, 228, 229, 663 (K.A.)
Smithers, D. W., with Dixon, 786 (Hel.)
Smythe, C. V. & Reiner, L., 580 (S.S.)
Snijders, E. P., 330, 738 (Misc.)
——, Postmus, S. & Schüffner, W. A. P., 840 (Y.F.)
So, K., with Kanda, 18 (Oph.)
Sobolewa, R. M., with Isabolinski, Stratanowitsch, Riwkina & Moskalewa, T. A., 619 (Fev.)
Société des Nations. Organisation d'Hygiène, 162 (Mal.)
Soda, T., with Shimojo & Adachi, 37 (Chl.)
Soesilo, R., 170, 474, 696 (Mal.)
—— & Gilbert, A. P. W., 474, 713 (Mal.)
Soetomo & Soetopo, 260, 865 (Lep.)
Soetopo, with Soetomo, 260, 865 (Lep.)
Sokolovski, W., 781 (Hel.)
Solowiow, N. N., with Kritschewski, 627 (Fev.)
Soltero, A. G., (825) (Misc.)
Sonnenschein, C., with Mayer & Laas, 234 (K.A.)
Sonoda, T., 781 (Hel.)
Soper, F. L., 834 (Y.F.)
—— & de Andrade, A., 78 (Y.F.)

Soper, F. L., Rickard, E. R. & Crawford, P. J., 836(Y.F.)
—— & Serafim, J., Jr., 502 (Y.F.)
——, Penna, H., Cardoso, E., Serafim, J., Jr., Frobisher, M., Jr. & Pinheiro, J., 77 (Y.F.)
Soru, E. 895 (Chl.).
——, with Damboviceanu & Combiesco, 894 (Chl.)
——, with ——, —— & Wisner, 893 (Chl.)
——, with Popesco-Combiesco, 894 (Chl.)
Souchard, 817 (Misc.)
—— & Roton, 8 (Lep.)
Soule, M. H. & Heyman, A. M., 285 (Dys.)
South African Medical Journal, 771 (Hel.)
de Souza-Araujo, H. C., 256, 264 (Lep.)
Sparrow, H., 240, 245, 247 (Fev.)
——, with Fejgin, 624 (Fev.)
——, with Mooser, 246 (Fev.)
——, with Nicolle, 627 (Fev.)
——, with —— & Giroud, 628, 629 (Fev.)
——, with —— & Laigret, 630 (Fev.)
Spector, B. K., with Tonney & Hoeft, (290) (Am.)
Spies, T. D., 25, 487, 490 (Pel.)
—— & DeWolf, H. F., 23 (Pel.)
Spyrou, 578 (S.S.)
Ssolowiewa, J. V., with Glusman & Predteschenskaia, 151 (Rab.)
Stamatesco, S., with Combiesco, Popesco & Angelesco, 245 (Fev.)
Stannus, H. S., 486, 489, 905 (Pel.)
—— & Gibson, C. R., 484 (Pel.)
Stefanopoulo, G. J., 842 (Y.F.)
——, with Pettit, 79, 838 (Y.F.)
Stein, A. A. & Karpichina, A., 538 (Lep.)
—— & Steperin, M. I., 550, (555) (Lep.)
Stein, L., 218 (S.S.)
Steiner, F., with Wigand, 106 (Hel.)
Steperin, M. I., with Stein, 550, (555) (Lep.)
Sternberg, E. J., 699 (Mal.)
——, with Kritschewski, (436) (Mal.)
Steudel, E., 201, 582 (S.S.)
Steward, J. S., 810 (Hel.)
Stewart, A. D., 37, 39 (Chl.)
—— & Moorthy, V. N., 49 (Ent.)
Stewart, F. H., 791 (Hel.)
Stewart, J., 389 (Hel.)
Stiles, C. W., 126 (Hel.)
Still, J., 85 (Y.F.)
Still, R. M. L. & Dang, M. L., (655) (Am.)
Stinnesberg, M., 823 (Misc.)
Stockman, R. & Johnston, J. M., 23 (Pel.)
Stoker, W. J., with Kuipers, 414 (Mal.)
Stone, A., with Bishopp & Cory, (72) (Ent.)
Stott, H., 181 (Mal.)
Strahan, J. H., 806 (Hel.)
Stratanowitsch, N. J., with Isabolinski, Sobolewa, Riwkina & Moskalewa, 619 (Fev.)
Stratman-Thomas, W. K., with Boyd, 165, 166, 688, 701 (Mal.)
Streef, G. M., with Radsma & Klerks (362) *bis*, 669 *bis* (Misc.)
Strickland, C., 30 (Pl.)
—— & Roy, D. N., 178 (Mal.)
Strong, R. P., (290) (Am.)
Strong, W. M., (825) (Misc.)
Struthers, E. B., Chang, H. H., Lin, L. C. & Ch'en, J. T., 662 (K.A.)
Stuart, G., 716 (Mal.)
——, with Getzowa & Krikorian, 150 (Rab.)
Sturton, S. D., 1 (Lep.)
Subrahmanyam, C., with Tull, 786 (Hel.)
Such Sanchiz, M., 9 (Lep.)
Sullivan, E. A., (29) (Pel.)
Sulmann, F., 313 (Chl.)
Sumerlin, H. S., (655) (Am.)
Sundar Rao, S., with Chopra & Mukherjee, 808 (Hel.)
Sundaram, S. K., with Mudaliyar & Ramachandran, 230 (K.A.)
Suzuki, K., 627 (Fev.)
Sweet, W. C., (475) (Mal.)
Swellengrebel, N. H., 162 *ter*, 710, 714 (Mal.)
——, with Cantacuzène, Ottolenghi & Pittaluga, 162 (Mal.)
——, with Nijkamp, 710 (Mal.)
Swynnerton, C. F. M., 67 (Ent.)
Symes, C. B., with Kauntze, 155 (Mal.)
v. Szathmáry, J., 899 (Chl.)
Szentkirályi, Z., with Lörincz, 235 (K.A.)

T

Takahashi, K. & Kodama, M., 242 (Fev.)
Talbot, 402 (Oph.)
Talbott, J. H. & Michelsen, J., 191 (H.S.)
Taliaferro, L. G., 706 (Mal.)
Taliaferro, W. H., 706 (Mal.), 818 (Misc.)
Tamiya, T., with Nagayo, Hazato & Mitamura, 242 (Fev.)
Tanaka, K., (252) (Fev.)
Tanon & Neveu, R., 72 (Ent.)
Tanturi, C. A., (400) (Hel.)
Tara, S., 433 (Mal.)
Tarassow, V., with Petruschewsky, 121 (Hel.)
Tarassow, W., 783 (Hel.)
Tareev, E., Epstein, E. & Gontaewa, A., 703 (Mal.)
——, ——, Pikul, I., Gontaeva, A. & Raskin, A., 698 (Mal.)
Tarejew, E., Epstein, H., Gantaeva, A., Raskin & Bolotina, A., 174 (Mal.)
Tarejew, E. M. & Poutchkova, A. I., 458 (Mal.)
Tate, P. & Vincent, M., 434 (Mal.)
Tavares, A. S., (483) (Bb.)
Taylor, 37 (Chl.)
Taylor, A. W., 846 (Y.F.)
——, with Lloyd, Lester & Thornewill, 66 (Ent.)
Taylor, E. L., 377 (Hel.)
Tchang, J., with Gajdos, 629 (Fev.)
Tedeschi, C. & Scalas, A., 415 (Mal.)
—— & Tortorano, L., (362) (Misc.)
Teglbjaerg, E. & Teglbjaerg, H. P. S., 24 (Pel.)
Teichler, G., 815 (Misc.)
Tejera, E., with Hill, 380 (Hel.)
Ter-Matevossian, C. M., 279 (Am.)
—— & Tsaturian, A. T., 289 (Dys.)
Thaysen, T. E. H., 22 (Pel.)
Theiler, M., 82 (Y.F.)
Theodor, O., (825) (Misc.)
Theodore, J. H., with Cunningham & Fraser, 847 (R.F.)
van Thiel, P. H., 426, 722 *bis* (Mal.)
——, with Essed, 792 (Hel.)

Thiry, U., 95 (Lept.)
Thewlis, M. W., (266) (Lep.)
Thomas, A. D., with Neitz, 149 (Rab.)
Thompson, W. E., 135 (Hel.)
Thomson, J. G., 353 (Misc.)
Thornewill, A. S., with Lloyd, Lester & Taylor, 66 (Ent.)
Thygeson, P., 14, 16, 401 (Oph.)
——, with Lumbroso, 401 (Oph.)
Tilli, P., 189 (Mal.)
Tirouvanziam & Phleng, (727) (Mal.)
Tisseuil, J., 6, 546 (Lep.)
—— & Berny, P., 265 (Lep.)
—— & Chorine, V., 554 (Lep.)
—— & Gillier, R., 265 (Lep.)
——, with ——, 555 (Lep.)
Toda, T. & Hiroki, H., 510 (R.F.)
Tolentino, J. C., with Rodriguez & Mabalay, 265, (874) (Lep.)
Tómasson, H., 477 (Bb.)
Tomb, J. W., 7 (Lep.)
—— & Helmy, M. M., 109 (Hel)
Tomikawa, R., 275 (Y. & S.)
Tonney, F. O., Hoeft, G. L. & Spector, B.K., (290) (Am.)
Topacio, T., 215 (S.S.)
Töppich, G., 789 (Hel.)
Tortorano, L. with Tedeschi, (362) (Misc.).
Toullec & Goiran, (483) (Bb.)
—— & Moreau, (475) (Mal.)
Toumanoff, C., 55 *bis* (Ent.), 155, 414, 466 *bis* (Mal.)
——, with Mesnard, 717 (Mal.)
——, with Morin & Moreau, (190) (Mal.)
——, with Robin, 701 (Mal.)
Toumansky, V., with Poljak, 31 (Pl.)
Tournier, E., 683 (Mal.)
Trabaud, J., (290) (Am.)
Trabut, G., 358 (Misc.), 862 (Oph.)
Tran-van-Don, (727) (Mal.)
Tran-van-Hanh, with Montel, Nguyen-van-Khai, Le-van-Phung, Truong-van-Que & Nguyen-Ngoc-Nhuan, 869 (Lep.)
Tran-Van-Nguu, with Guy, 14 (Oph.)
Trapesontzewa, C. & Nikolskaja, Z., 403 (Oph.)
Traub, E., 639 (Rab.)
Travassos, J., with Monteiro, (504) (Y.F.)
Trawiński, A. & Maternowska, I., 802 (Hel.)
Treillard, M., 718 (Mal.)
——, with Roubaud, 715, 718 (Mal.)
Trensz, F., 57 (Ent.), 705 (Mal.)
Tretjak, with Afanassiewa, 238 (Fev.)
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